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PERSONAL RECOLLECTIONS OF GRAEFE, ARLT AND DONDERS*

HASKET DERBY, M.D.

BOSTON

EDITED BY GEORGE S. DERBY

The birth of modern ophthalmology may be said to date from the invention of the ophthalmoscope, the news of which Helmholtz first published in 1851. The beginnings of the new science were chronicled in the *Archiv für Ophthalmologie,* brought out in January, 1854, under the direction of Albrecht von Graefe who was entirely responsible for the first half of volume 1. The second half of the first volume appeared in April, 1855, and the names of Ferdinand Arlt in Prague and Franz Cornelius Donders in Utrecht were thenceforth associated with that of the illustrious founder as editors. The triumvirate remained unbroken until the death of von Graefe in 1870.

It was in the winter of 1859 that I became a pupil of Arlt. The following year I passed with von Graefe in Berlin. I went from there to Utrecht, and later attended the clinics of Bowman and Critchett in London and of Sichel and Desmarres in Paris. Daily contact with those masters of the science made me familiar with their personal peculiarities, plan of teaching and methods of practice. There are but few now living who had the privilege of observing and sitting at the feet of these great men, while they were yet in their prime. And the few must soon, in the course of nature, pass away. Before it is too late I have thought it might be well worth while to rake over the ashes of memory, and strive to bring the present generation face to face with those whose teachings are as familiar to them as their faces are obscure. After a great leader in science has been gathered to his fathers, time often invests him with a halo that blurs his lineaments. The voice may still resound, but the manner of man is no longer apparent.

My own recollections of those early days are still clear and bright. It was during the winter of 1859, while pursuing the study of general medicine at Vienna, that I felt I should acquire some knowledge of diseases of the eye. I was fitting myself to be a family physician, and wished to be able to give treatment in the ordinary cases that might occur in my own practice.

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I had never seen an ophthalmoscope, and it was necessary to learn its use. Two courses were given, one by Arlt's assistant, the other by Prof. Eduard Jaeger. In the former, the use of the inverted image with the small Liebreich mirror was taught exclusively, while Jaeger preferred and taught the direct method. The instrument he himself had invented contained two mirrors, the "licht stark" or silvered concave, and the "licht schwach," a modification of the one originally proposed by Helmholz, three plates of unsilvered glass superposed. It was by the use of the upright image that he made the superb ophthalmoscopic atlas, the drawings in which were executed by his own hand and printed in color at the Imperial printing office of Vienna in 1855. This work, often imitated, has never been surpassed, and retains its authority to the present day.

Fig. 1.—Hermann Helmholz.

Jaeger came of good stock. He was the son of Friedrich Jaeger who had long occupied the highest rank as an oculist as well as a general practitioner in Vienna, and who was still living in my day; and the grandson of Beer, the first professor of ophthalmology at the university. He was a kindly man, then approaching middle age, a most excellent and painstaking teacher of the ophthalmoscope, but a somewhat dull and erratic lecturer. I have full notes of some of his lectures, but they would be devoid of interest at the present day. While looking them through not long since, I came across one which, though unusually disconnected, amused his hearers so much that I cannot resist the temptation of giving its skeleton. I took it down exactly as it was delivered. As one of the objects of this paper is the illustration of the personal ways of the teachers of those days, I have less hesitation in laying it before you, promising that I have no intention of holding up to ridicule one whose ways were gentle and whose disposition was kindly.

8:15 a. m.: On entering the lecture room I find but two men present, and Jaeger holding forth to them with great rapidity. He successively adverts to military surgery, presbyopia, himself and the weather.

8:20: He suddenly pulls up and inquires if he expressed himself clearly and distinctly in his last lecture. The black-bearded man in the gold spectacles chuckles and nods. (This man, by the way, puts the title of prince on his visiting card and always ties a red bandanna handkerchief over one eye when endeavoring to use the ophthalmoscope.)

8:25: Jaeger is uncertain where to begin, but finally starts off at a breakneck pace on the evils attendant on the formation of a white spot in the macular region.

Fig. 2.—Eduard Jaeger.

8:35: I had drowsed away but on coming to find the professor eagerly discussing retinal separation. Three minutes later he ties himself up in a hard knot, and stops short with a wild look, apparently in the middle of his subject. He rubs his chin (a favorite gesture), smiles benignly and diverges to tubercles in the choroid.

8:45: He is talking about microscopic preparations in general and the length of time he has had his own. General stampede about the room to see the specimens.

8:50: After a silence of one minute, during which he has looked the class smilingly in the face, he comes to himself and mildly inquires what he was last talking about. Being informed, he begins on scleroticochoroiditis posterior.

8:55: He is discussing goggling.

9:00: He has diverged to curve of cornea.

9:10: He is running about the room, looking for chalk.

9:15: He unexpectedly stops.

Despite his eccentricities, he would have attained more prominence in his day had he not been so completely overshadowed by Arlt, who was then the regular professor, while Jaeger occupied the subordinate position of "extraordinary professor."

It was at his "operation course" that I first made the acquaintance of Arlt. It was given daily at noon in a room used for the Poliklinik, and was confined to about a dozen participants, all graduates in medicine and most of them foreigners. My name having been inscribed on the list some time in advance and accepted, I found myself, at the appointed hour, in the presence of an insignificant looking man, rather below medium height, somewhat shabbily dressed in black, with sharp cut features, a large and suspiciously red nose; he was wearing a high stock

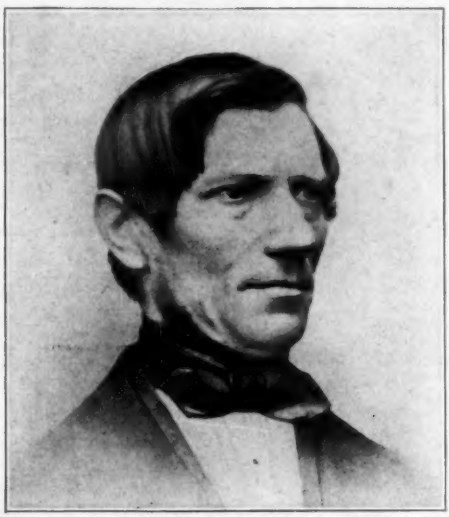

Fig. 3.—Ferdinand Arlt.

of black satin and showed no collar. The latter, in fact, he never donned except on state occasions. His general appearance was so insignificant and ordinary one of the class observed, on seeing him riding in his carriage, sitting up straight and perched on the extreme front edge of the seat, "he looks as if he had been sent for a carriage by someone else and were bringing it home to him." How his appearance belied him! He was the first of living operators, the most thorough and able of teachers, and a man of worldwide fame. Patients came to him from all over Europe. His "Klinik" was thronged by physicians of every clime. He was not a commanding genius like Graefe, but a born teacher and a self-made man of vast learning and originality. Graefe himself said to his students, "If you would learn to operate, go to Arlt. In comparison with him I can do little for you."

During the first part of the course, the material consisted of pigs' eyes inserted into a wooden mask. On these we learned iridectomy and the various operations for cataract, discission, reclination and extraction. The latter was the old flap extraction, done with the triangular Beer's knife. It was with infinite care that Arlt explained the proper manner of holding this knife, as well as the exclusively wrist motion with which the cut was to be completed, the arm being held immovable. Graefe's operation for extraction had not yet been given to the world. Enucleation, tenotomy and the operations on the lid were taught with the human head, a fresh one being procured daily from the dissecting room. Nothing was left to an assistant; the professor himself stood over and directed each student and saw that each performed every operation a specified number of times, keeping a journal for the purpose. We were made to operate with either hand, as for instance with the left in the case of cataract of the right eye, the flap being made downward.

No Prussian martinet ever watched his drill more closely than did Arlt his class at their operations. Far and wide, he himself was renowned for his dexterity as an operator, surpassing in this respect all his contemporaries. How well do I remember seeing him perform an extraction at the "Klinik" of Dr. Röder at Heidelberg, during one of the meetings of the Ophthalmologische Gesellschaft. Giraud-Toulon, the well known French savant, was present, and as Arlt took the knife in his hand, he turned to a colleague and I heard him say, "Maintenant vous allez voir quelque chose de classique."

Every medical student intending to take a degree was obliged to take and report on a case in the wards. At the bedside, Arlt was a severe master. He would question the pupil in the most searching manner, requiring a thorough knowledge of the nature and possible outcome of the disease, and never excusing negligence or forgetfulness of instructions already given. But it was from his treatment of the outpatients in the Poliklinik that we learned the most. He came there after his morning visit in the wards, and remained from 10 till 12 o'clock. My comrade, Dyer, and I always slipped away from the ward a quarter of an hour before the conclusion of the visit in order to secure places near the professor's chair. Theoretical lectures were almost never given; neither were the patients selected or classified. They came up one by one, and their condition was then and there diagnosed, commented on and prescribed for. Cases of refraction seemed comparatively rare in those early days; the few patients who came for that purpose were sent into a side room, where we never strayed, and were prescribed for by an assistant. Astigmatism was then unknown, and the choice of glasses by the surgeon rather than by the optician was only beginning to be practiced. Deep-seated disease was seldom brought to our notice. Diseases of the lids, conjunctiva, cornea and iris, how-

ever, were demonstrated and explained in a manner that I have never seen surpassed. The connection between the state of the general health and these local conditions, and the necessity for a proper diet and a good hygienic environment were insisted on as by no other teacher with whom I came in contact. In these respects, the instruction given by Arlt has been of the greatest value to me during my whole professional career.

The professor was of a most kindly and lovable disposition, a child of nature. One day, several of us were dining at the Riedhof, the principal restaurant of our quarter, when Arlt unexpectedly came in and seated himself in our midst. At the close of the meal he selected for his dessert "Krapfen mit Saft," three little fritters floating in a sweet red sauce. Two of these he finished, but he had already dined too well to absorb the third. Not desiring to waste this delicacy he cast his eyes about him; then, with his own knife and fork, he lifted the surviving "Krapfen" from his own plate and, with a bow and a smile, deposited it on mine which happened to be empty. He was conferring a distinction, paying a great compliment. Dear old man! I ate the Krapfen to the last crumb.

He was charitable to a fault, often assisting the needy from his own means, and most considerate of the feelings of others. I saw a shabby, genteel person, with a certain air of having seen better days, once seat himself before Arlt at the Poliklinik. "Your business?" inquired the clerk. "Actor," was the reply. The clerk, as in duty bound, was going on to inquire the name and residence, but Arlt nudged him to desist, spoke kindly to the man, prescribed for and dismissed him. Struck by the purity of the actor's accent I followed him out to see whether I could not get him to give me some lessons in pronunciation. I thus made his acquaintance and, later, that of his family, who were most estimable people, in sadly reduced circumstances. The incident showed, on the part of our teacher, a kindly sympathy with honest poverty that I have never forgotten.

The simple surroundings of Arlt's early days never failed to affect his subsequent life. Born in a little Bohemian village near Teplitz, in 1812, his father a blacksmith, his mother the daughter of a miner, he lived at first among the humble peasants who cultivated their little patches of ground and worked in the mines. The wages of a miner in those days was about 10 cents of our money, and meat was used at the Sunday dinner only. In Arlt's reminiscences he gives an anecdote of his youth which is worth repeating, and which I relate in his own words: "I might have been 7 years old when one day my mother told me to get a pot of blood on my way back from school. When a calf is slaughtered the blood is collected and stirred quickly with a rod, before it has had time to coagulate. It is thus converted into a fluid of uniform density;

into this, pieces of bread are dipped, the whole put into a pan and baked, forming a kind of sausage. It was a cold winter's day, and as I was carrying my pot past one of the upper houses of the village I slipped on the ice; away went the blood all over my leather breeches. The wife of one of the villagers took pity on the sobbing lad, made me come into her house, and sent word home by one of the other school children that I would spend the night with her. Forty years later I operated on the same woman for cataract, and made her happy by enabling her to read her prayer-book."

Aided by friends and partly supporting himself by instruction given to his fellow scholars, and living on a diet of soup, bread and potatoes, he struggled through the gymnasium at Leitmeritz. Later he obtained an appointment as a private tutor, and saved enough to enable him to enter the University of Prague as a student of medicine. It would take too long to detail all the events in his subsequent career: his becoming the assistant of Professor Fischer, his success in Prague in ophthalmic practice, his obtaining a position at the university, first as instructor and later as professor, his appointment to Vienna on the death of Rosas, and then, on his appointment as co-editor of the *Archiv für Ophthalmologie* with his old friend, Graefe, his rise to European distinction.

All this may be found in his autobiography, which he wrote after enforced retirement from his chair at the age of 71, and which was published in 1887, with an account of his last illness by his former pupil, Otto Becker. Arlt died in March, 1887, after a protracted and most painful illness. Of all my teachers, none was ever kinder to me, and no one did I regard with such personal affection.

On reaching the age of 70, Arlt notified the Ministry of Instruction that, in accordance with the law, he was ready to leave his professorship. The minister, however, paid him the compliment of inviting him to remain in office a year longer, as he still was well and strong, and his actual retirement took place twelve months later.

It was on this occasion of his reaching his seventieth birthday in 1882 that his pupils, the world over, united in offering their beloved master a token of their regard. Each sent his photograph to a committee which had been formed in Vienna, together with a contribution toward the purchase of a suitable album, a design for the cover of which was to be made by a competent artist. When the album was completed, it was presented to the professor, who says in his autobiography, "A supreme distinction, an occasion of the greatest joy for me, was the celebration of my seventieth birthday by my former assistants and pupils. An album designed by Stork, who gave the drawing for a suitable cover, and which contained some two hundred photographs of my old pupils, was presented me and forms a most treasured keepsake for myself and my children."

Arlt showed me the album the last time I saw him in Vienna. To each of the subscribers was sent a copy of the cover. The design was suggested by the passage in the "Scriptural Book of Tobit," in which the young Tobias, attended by the angel Raphael and accompanied by his faithful dog, restores his blind father to sight.

On leaving Vienna the professor was kind enough to give Dyer and myself a letter of introduction to Graefe. On our arrival in Berlin, in the fall of 1860, we called on Graefe one morning to present it. I shall never forget the aspect of the room in which we first met the great man. He was as yet unmarried, which perhaps accounted for the disorder of his surroundings. Sitting in his library, at a table filled to over-

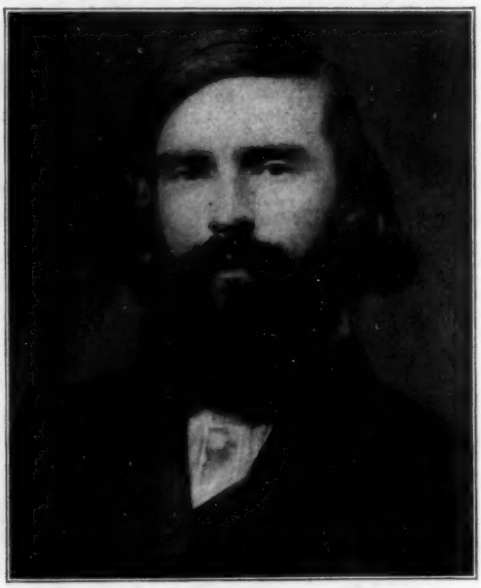

Fig. 4.—Albrecht von Graefe.

flowing with books, pamphlets and manuscripts, a corner having been hastily cleared away for the tray that held his breakfast, he was having his morning meal of bread and coffee, while dictating to his secretary. Trifles impress one at such a moment; I well remember seeing on a sofa a pile of new hats, still in their wrappings. Evidently he had had no time to go to the hatter and intended to make his selection at home. He received us graciously, but he made a wry face as he thought of his duties to his class, so soon to be assumed. "There's an end to my fine morning hours," he said ruefully, addressing his secretary. He read our letter and welcomed us to Berlin. As we took our departure he called us back and said, "You had better enroll yourselves as praktikants;

that's the best way to learn." Without knowing precisely what this meant, we went round to the "Klinik" the same day and put down our names as he had advised.

Graefe was then 31 years of age and at the pinnacle of his fame. The *Archiv* had been founded six years before, and the great discovery of iridectomy for glaucoma was but three years old. Patients from every corner of the world thronged to Berlin. The "Klinik" in the Karlstrasse was beset by the poor in its lower stories, and thronged

Fig. 5.—Von Graefe's Klinik in the Karlstrasse.

with the rich in the private rooms above. The working capacity of the professor was unbounded. He was not as yet full professor at the university; old Jüngken clung like a leech to the office, and Graefe was but a subordinate and had had to establish his own private hospital. At 7 o'clock in the morning he was at his desk in his private residence, answering letters, writing for the *Archiv* or engaged in study. His own hospital work was done at a large building in the Karlstrasse, the whole of which he occupied. The reception, lecture and operating rooms were in the lower story, the wards and private rooms above. Six mornings

in the week he would come here at 9 o'clock, giving a theoretical lecture on three days and holding a clinical conference on the others.

Patients suitable for demonstration and forming good material for clinical study were selected by the chef de clinique and came at 8:30 on conference days. At the same hour, the praktikants came, volunteers from the large class that was regularly in attendance. There were rarely more than six or eight of us. To each was assigned a patient he had never seen before, and he was allowed half an hour for examination, being expected to form a diagnosis and prognosis and advise a plan of treatment. At 9 o'clock, the professor took his seat, the first patient was called in and placed in a chair in front of him and the praktikant sat down in a chair at Graefe's side. The class, from 60 to 100 in number, stood or sat around. The man in charge of the case was closely questioned, made to give the reasons for his conclusions, and often to draw on the board the appearances that he had observed. The anxiety experienced on these occasions was intense. Between the difficulties of the language and my own ignorance on ophthalmic subjects, as taught in the school of the great master, I made so many blunders that I sometimes wished the floor would open and swallow me. But the moments were golden and the instruction given was never to be forgotten; indeed, it seems to ring in my ears as freshly today as it did forty-five years ago.

After the conference was over, the professor would visit the Charité Hospital, his round in the city, take a hasty dinner, often snatched in his carriage, and at 2 or 3 p. m. return to his hospital for his operations, often twenty or thirty and embracing every variety of ophthalmic surgery. At these, each member of the class could be present but once a week; we were divided into three sections for this purpose. The poliklinik followed, from 100 to 200 poor patients being admitted, prescribed for, and if necessary and space permitted, retained in the house for operation or treatment. Graefe made a hasty visit through the wards of his extensive establishment, and then drove rapidly home to hold his own private office hours. These commenced at 7 o'clock in the evening and might last till late into the night, from fifty to seventy patients often being prescribed for. When they were gone Graefe would return to his scientific labors, interrupted in the morning, and continue them till forced to seek repose.

He was a strikingly handsome man, tall and rather thin, with long flowing hair, a full beard and regular, classic features. The Germans said he had a Christuskopf; it was indeed a noble countenance and resembled some of the portraits of the Savior as painted by great artists. His manner was magnetic, his intelligence most keen, and his argument clear, lucid and convincing. When he lectured he was undertaking no perfunctory task; there was no droning digest of already accepted

facts and principles. What he said was often not to be found in books or in the teachings of others. One felt that the information was being communicated to him at first hand and for the first time. One could have listened for hours.

Besides his chef de clinique, a young physician, Graefe had six assistants, all being of nearly the same age as himself and selected from among his personal friends: Arendt and Ewers, who attended principally to the private patients; Michaelis who hovered round generally but whose duties I never clearly understood; Schweigger, still living,

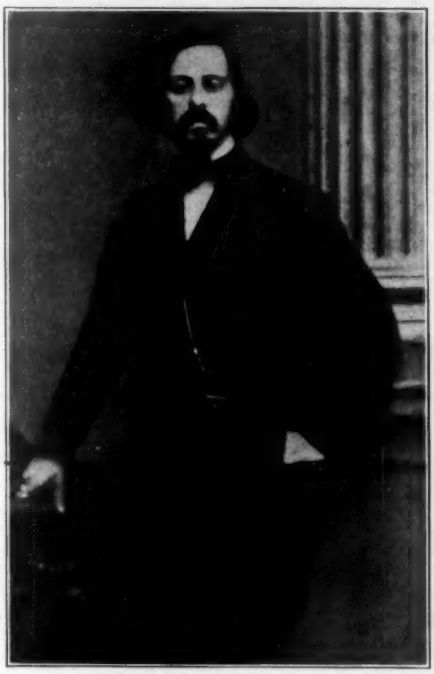

Fig. 6.—Oscar Liebreich.

who made the pathologic studies and gave courses on microscopy; Schuft (who afterward changed his name to Waldau) who assisted at operations and gave most of the operative course, and last, but by no means least, Liebreich, also still alive. He was by far the brightest man on the staff; performed all the ophthalmoscopic and refractive work and gave the ophthalmoscopic courses. Rather short and thin, with long hair and sharply cut semitic features, and always clad in a dress suit, he exactly resembled a dancing master temporarily deprived of his fiddle; he was said to be an artist of no mean ability. Of his preeminent skill with the ophthalmoscope it is unnecessary for me to speak; his atlas tells the story. He did the refractive work at the Klinik, using the old Jaeger

tests which were then universally employed, as it was before the day of Snellen. He had an old mahogany case of lenses, all based on the scale of the Prussian inch, and their values expressed in vulgar fractions. These glasses had no setting; they were simple lenses, and were hastily inserted in wire clips, the distance between the eyes being invariable. I can well remember a familiar gesture of Graefe's. Just before inserting a lens in the clip he invariably polished it against the silken lining of the left lower flap of his coat, which he pulled forward for the purpose.

How some little things linger in the memory after long years have passed! At the Klinik, we kept a large buck rabbit for ophthalmoscopic practice. This creature would be placed on a chair, near a powerful lamp, and a student, kneeling on either side, would examine the fundus, the animal remaining perfectly still, but with his sides quivering in sugpressed emotion. He was known familiarly as "der alte Stammhalter," and had been a long time in the service. One day the Stammhalter disappeared and his loss was much bewailed. As is the case with every similar institution, the Klinik had in its employ a man who had grown up with it and formed an integral part of the establishment, combining the duties of janitor, superintendent and general detective. Such was our Andreas, a tall, rawboned, athletic Berliner. I never knew his last name. Andreas went to market one day; there, in the part devoted to live animals, he found a large rabbit whose appearance was strangely familiar. To prove property, he rushed back and brought Liebreich who then and there examined the rabbit with the ophthalmoscope and recognized certain peculiarities about the optic entrance. It was indeed the Stammhalter, and he was brought home in triumph.

Speaking of Andreas reminds me of Graefe's method of dealing with crying children. Nothing annoyed him more than a bawling boy; he would instantly give a sign to Andreas, who brought in a basin of water, took the child up deftly, pinning his arms and legs, and inserted his head in the basin. On his withdrawal, the patient was made to climb up on the seat of a chair and then instantly to jump off. This process was repeated over and over again, until out of breath and fairly cowed the child no longer had energy enough to yell. Graefe was a bachelor at that time, but later married and had children of his own. I suspect that he did not carry this method into practice in his own family.

Another incident at the Klinik that I have never forgotten seems to have occurred but yesterday. Graefe sits at his table, his clerk by his side. He has just sent Liebreich with a patient into the little dark ophthalmoscopic chamber to the left, and is himself examining a new patient. Out from behind the curtain comes the thin, piping voice of Liebreich, uttering a single word, "cysticercus." Up starts Graefe as if he were shot, almost upsetting the table before him. "Donnerwetter!"

he exclaims, "das muss ich sehen," and disappears in the direction of the voice.

Graefe invariably examined in the inverted image and used a so-called Coccius ophthalmoscope, a square plane mirror with a convex lens of short focus, attached to it by an arm, at an angle of 45 degrees, bringing the rays from the lamp to a focus on its surface. I never saw him use any other. Liebreich employed the small concave mirror with a clip behind it. He, too, seldom examined in the upright image. There was one amusing point of difference between him and Jaeger. The latter insisted that there was nothing objectively visible about the macula lutea, Liebreich that there was.

I was present at the meeting of the Heidelberg Ophthalmologische Gesellschaft when Graefe, for the first time, presented his new operation for the extraction of senile cataract. Both his presence and his paper were greeted with enthusiasm. He showed us the long narrow knife which he used in its performance, the only thing about the operation that has survived its inventor.

This was, I think, in 1865, and he was apparently as well and bright as ever. At the meeting of 1868, however, he was sadly changed, having grown gray and bent, and also somewhat emaciated. But the old spirit survived. Graefe was full of fun and had a keen sense of humor. When in Paris he used to go out to Bellevue and stay with Lüer who told me that the professor wanted no serious people around; he asked Lüer to invite only "heitere Leute" to meet him. At the dinner given that year by Knapp at the Old Castle, just before his departure for America, it was my privilege to sit at the side of our illustrious master and listen to his genial and friendly words. I well remember how, on this occasion, he chuckled at the idea advanced by one of the speakers that one of the perils to be encountered by our friend, Knapp, among the savage tribes who were understood to infest the United States was that they might esteem him a succulent morsel for one of their repasts.

He failed steadily during the next year, the victim of phthisis. In the spring he performed his last operation, one of importance. He was lifted from his carriage, carried in a chair to the bedside, and did his work with a trembling hand. In July of that year, 1870, I was in Berlin and called on him at his residence. Waiting outside the door of his study I heard his voice for the last time, as he told his secretary that he was too ill to see any one. It was but a few days before his death.

"July 20th," to use the words of another, "was one of the few warm days of a cold and rainy summer. During the evening hours Graefe sought coolness and refreshment in the airy conservatory attached to his beautiful house. That he might pass a comfortable night his bed was prepared in the same place. With a lessened sense of oppression, and perhaps less occupied with the thoughts of death than for a long

time, he cast himself at midnight on his couch, suddenly as was his wont. Then came on the sore distress that was to abruptly extinguish this great life." Traube, his friend and physician, was hastily summoned. "Es ist die Agonie," he sadly said. "Graefe died on the night of the twentieth of July, amid the noise and confusion attending the outbreak of a terrible war." My brother, who at the time was one of his assistants, was present at the funeral. He said it was the largest that he ever attended. The Victoria Strasse, where Graefe lived, and all the neighboring streets, wherever there was standing room, were filled with deputations from the city, students with their corps and banners, and equipages from the Court with footmen in livery of royalty. The faculty of the university, chamberlains from the Court, and many per-

Fig. 7.—Franz Cornelius Donders.

sons of distinction who had known and loved him, surrounded the catafalque.

He was but 42 years of age when he died. What life of twice that length has more enriched ophthalmic science and thereby benefited humanity? Between the old "Augenklinik" and the Charité Hospital stands a memorial to Graefe, in bronze and stone. His own figure is life size, and he is represented in the act of descending a step from a chair he has occupied in a niche. On either side extends a wall of stone, and in the wall on each side is a bas-relief in bronze representing patients in different attitudes. I am sorry to say that the likeness of the great master is most imperfect. As the monument was a public one it was necessary to obtain the Emperor's assent to its erection. It is said that old Kaiser Wilhelm at first objected. "Der Kerl war nicht amal Soldat," said the grim old warrior.

In many respects ophthalmologists are personally more indebted to Donders than to the other members of the great triumvirate. To him is principally due the knowledge of refraction. Who can say but that nine tenths of his cases are cases of refraction, especially in private practice. Donders' was a very different personality from that of his colleagues. When I came to Utrecht in the fall of 1861, he was 42 years of age and in the prime of his powers.

Donders was a man of noble presence, above the average height, inclined to portliness, with a massive head, cleancut regular features and a piercing eye. His manner was grave and his words few, but his native kindliness shone in every act. He greeted us, two insignificant, unknown students, with the utmost cordiality; he at once took us around to the reading room where he put down our names, and taught us our first lesson in Dutch by instructing us how to ask in that language for the English papers. His interest was even more strongly in evidence the next day, when we first made our appearance at his morning lecture. We had expected to understand but little of it, but to our delight he informed the class that as there were strangers present who understood no Dutch, and as they themselves were all familiar with German, he would for the present carry on the lectures in the latter language. This he did during our entire stay.

Except at these morning lectures and at his comparatively infrequent operations, we saw little of Donders personally. He was then hard at work studying astigmatism, but had published none of his observations. These first made their appearance in 1862, when he brought out the brochure "astigmatism and cylindrical glasses," in Dutch and German, with a touching dedication to his friend Graefe who had recently recovered from a dangerous illness. "I have been irresistibly impelled," he said, "to dedicate this work to you, my noble friend. . . . Let it be to you a slight proof of my great joy, and bring you from the depths of my heart congratulations on the dawning spring of your new life."

It was at Utrecht that we made the acquaintance of Donders' chief assistant, Snellen, whom I have found a life-long friend. We observed his work at the "Klinik," which he managed for the professor. The cases were relatively few, but they were studied thoroughly. In the afternoons we worked in the laboratory at measurements of the corneal surface, using the ophthalmometer of Helmholtz, then a recent invention. One of our companions was Doijer, later a professor at Leyden. There were a few foreign and Dutch physicians attending the hospital. Among them was Haffmans who wrote a monograph on glaucoma that in its day attracted much attention.

I met Donders for the last time at Heidelberg in 1888, when he presided over the Congress. On the evening of the first day he took a walk with Leber, and invited me to join them. They discussed the

management of the *Archiv*, of which they both were editors. Although he had already reached the age of 70, Donders never seemed in better health; his eye was not dim, nor his natural force abated. In less than a year there came the shocking news of his death, which took place on March 23, 1889. The last of the giants was gone.

Beyond a wealth of material, London in the early sixties offered little to the student of ophthalmology. The Moorfields Hospital of that day, thronged as it was with patients, had little in common with the magnificent and perfectly appointed establishment that now has taken its place. We found a rude form of ophthalmoscope in use, a sort of miniature shaving glass with no clip behind to hold a correcting lens.

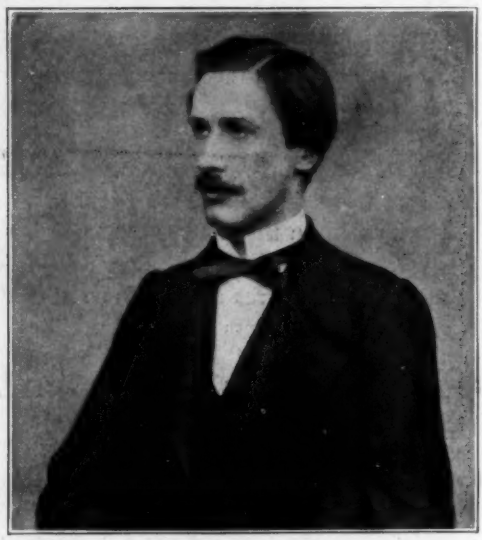

Fig. 8.—Herman Snellen.

I well remember the indignation of one Indian surgeon who was examining a case ophthalmoscopically, when I cautioned him to beware of falling through the hole in the center of his mirror. Refraction was hardly ever practiced, although it was beginning to be studied eagerly.

Finding that we had been students with Graefe and Donders, almost all the Moorfields surgeons allowed Dyer and myself, during our stay in London, to examine and prescribe for all the patients requiring refraction who attended the hospital, and they accepted our diagnosis and treatment. Truly did we feel then that in the kingdom of the blind the one-eyed is king. Besides Bowman and Critchett the attending surgeons were Wordsworth, Streatfield, Hutchinson, Hulke, Couper, Poland and Lawson. Dixon and Bader were also on the staff. Interested in many other departments of medicine, none of these men had been able to give

exclusive attention to ophthalmology, as was, indeed, apparent. They were all most kind to us, many showing personal hospitality. Of the whole number, Hutchinson and Couper alone survive. With the sole exception of Arlt no more finished and dextrous operators than Bowman and Critchett were to be found on the Continent; witnessing their manipulations was in itself a liberal education. Bowman's removal of cataract was remarkable for its elegance. The vicious operation of iridodesis, which was unhappily then in vogue, was performed by Critchett with a grace and ease worthy of being employed in a better

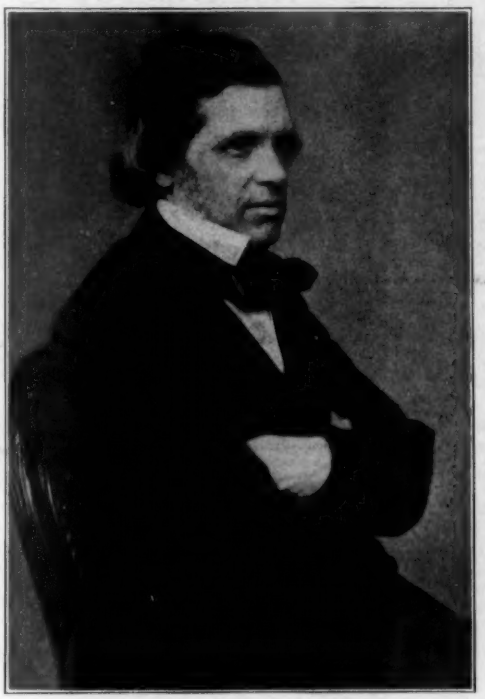

Fig. 9.—Sir William Bowman.

cause. Tying a fine silk thread around a prolapsed iris is by no means as easy as it appears to be in the hands of a master.

We had to go to Paris to provide ourselves with instruments, and while there saw the clinics of Sichel and Desmarres. The former was in his day a leading light and had written various learned works, but his sun was setting. I found him a little old man, of a distinctly Hebrew cast of countenance. As is apparent from his name, he was of German origin, being known in his own country as "der alte Sichel." His practice had grown small, and his cases for demonstration were few. He

was impatient of modern theories and modern methods of investigation. With the naked eye he had discovered two or three synechiae in a case of iritis, and was greatly annoyed with me for making out several more by oblique illumination. A favorite expression with him was, "Dans ce cas là l'ophthalmoscope ne nous montre rien." It was most unfortunate that he should still be posing as a teacher.

Of Desmarres himself we saw but little, his clinic, then by far the largest in Paris, being conducted by his son Alphonse and his assistant Galezowski. The latter was most capable, but evidently felt his subordinate position. "Le papa Desmarres," as he was familiarly called, appeared but once or twice during my stay, so that I am not competent to pass any judgment on his abilities. He was a typical Frenchman, a

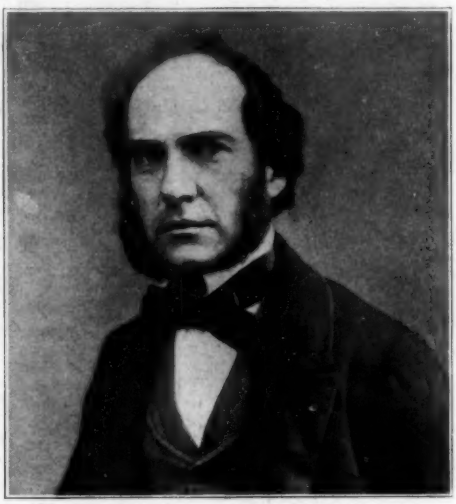

Fig. 10.—George Critchett.

big, burly fellow, brusqueness personified. He left behind him a work on the eye in three volumes, and is said to have been a great teacher. Graefe himself, during his stay in Paris, was his chief assistant.

After a few months in Paris, Dyer and I came home and engaged in practice for ourselves, he in Philadelphia and I in Boston. How much we thought we knew, how impossible seemed the likelihood of further advance in the knowledge of ophthalmology! And yet how great was our ignorance, as compared with the present day. We had no knowledge of asepsis. There was no local anesthesia. There was no known myotic. We were unaware that artificial dilatation of the pupil might bring on glaucoma. Patients with trachoma, then prevalent, were treated by daily touchings with silver nitrate and bluestone, year in and out, jequirity and expression being in the womb of the

future. The operation for senile cataract was still flap extraction with the triangular knife, and 85 per cent of success was considered a triumph of surgery. There was no ophthalmometer save the recently invented instrument of Helmholtz, costly, complicated and requiring mathematical knowledge. The trial cases, rudely constructed, contained no cylinders. Even after astigmatism became known, the cases were furnished with but one row of cylindric glasses. The first that contained a double row was made for me by Nachet, under protest. He could not see what I needed it for. Muscular insufficiencies were unknown; the prisms were few and clumsy. The blackboard was the only perimeter. The construction of the ophthalmoscope was clumsy and elementary, but few lenses coming with the instrument and these having to be inserted one by one in a clip swung back of the mirror. There was no shadow test. And so I might go on at length.

Nevertheless, the young man of that day, fresh from the instruction of Graefe, Arlt and Donders, felt that he represented the rising sun of science, and was even apt to believe that all knowledge was concentrated in his own person. With how much contempt did he regard his less traveled colleagues. Under such circumstances it required long years to acquire a proper modesty in regard to one's own real attainments, and to gain a fitting respect for the acquirements and knowledge of others who had either been brought up in a different school, or were forced to rely on the results of their own experience. Happy he who has learned the lesson betimes!

DOES KERATOMALACIA EXIST IN ADULTS?*

ARNOLD PILLAT, M.D.
PEIPING, CHINA

In spite of certain investigations published during the last decades in both Europe and America, the question of the existence of keratomalacia in adults has thus far been answered in the negative in the text books. This is to a certain extent justified by the fact that in former years many diseases which bore no relation to each other, such as neuroparalytic keratitis, senile marginal ulcers, senile marantic ulcers (Schoeler [1]) and keratomalacia, were all considered one disease. It is also due to the fact that the general high standard of living and the relatively well nourished condition of the population of Western and Central Europe and America have caused this disease to disappear there, or to appear in such isolated cases that, in view of the prevailing opinion that keratomalacia is exclusively a disease of childhood, it has been impossible to make the diagnosis of keratomalacia in adults. Reports on diseases similar to keratomalacia, accompanying serious conditions of the liver ("ophthalmia hepatica," Weiss;[2] Hori,[3] and others), have added to the confusion. The statement that keratomalacia is exclusively a disease of childhood (Fuchs-Salzmann [4]) therefore seems justified, and the following sentence expresses the whole uncertainty of the question: "Genuine keratomalacia does not appear in adults but it is not impossible that marantic corneal ulcers are analogous to keratomalacia." De Schweinitz [5] also stated: "Keratomalacia usually appears in infants during the first years of life" and added only that it was occasionally found in slightly older children. In examining the literature it will be found that reports on keratomalacia in children extend to the tenth year of life, but cases at this age are rare and are emphasized as such accordingly. As an exception, Heine,[6] in discussing keratoma-

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* From the Department of Ophthalmology, the Peiping Union Medical College.

1. Schoeler: Beitrag zu den xerotischen Hornhautleiden unter Aufführung einer neuen, bisher von mir nicht beobachteten Form dieses Leidens nach vorausgegangener Entziehungsdiät, Berl. klin. Wchnschr., 1887, p. 979.

2. Weiss: Berl. klin. Wchnschr., 1873, p. 232.

3. Hori: Zur Anatomie einer Ophthalmia hepatica, Arch. f. Augenh. **31:** 393, 1895.

4. Fuchs-Salzmann: Lehrbuch der Augenheilkunde, ed. 15, Vienna, Franz Deuticke, 1926.

5. De Schweinitz: Diseases of the Eye, ed. 10, Philadelphia, W. B. Saunders & Company, 1924.

6. Heine: Die Krankheiten des Auges im Zusammenhang mit der inneren Medizin und Kinderheilkunde, Berlin, Julius Springer, 1921, p. 61.

lacia in "The Diseases of the Eye in Connection with Internal Medicine and Pediatrics," mentioned that "in the era of steam, this (keratomalacia, generally) has become rare, but one extremely hot summer (year not stated) I observed a complete picture of hemeralopia with xerosis in the inmates of a poorhouse, which caused the loss of several eyes, after the exact manner of infantile keratomalacia." There is hardly any doubt, that this was an instance of keratomalacia in adults, but the observations were considered exceptional and were too vague to construct a clearly defined picture of the disease.

Elschnig [7] stated that keratomalacia occurred almost without exception in artificially fed and poorly nourished sucklings with intestinal diseases, rarely in older children and very rarely in adults. But the passage: "The cornea shows, especially in the marginal areas, deep, round infiltrates, which rapidly growing towards the center of the cornea . . ." is not correct at least of keratomalacia of adults in China, in which the ulceration of the cornea usually starts in the center.

In the early literature one finds isolated cases of keratomalacia in adults and similar conditions, always in connection with other diseases, (Groenouw [8]). Hildreth [9] was the first to report (1870) on keratomalacia in an adult, a woman, aged 52, who had had acute diarrhea for three months. The patient was undernourished and had lost a great deal of weight. "Hornhautverschwaerung" appeared a few days before death.

In 1866, Gama Lobo [10] reported the appearance of keratomalacia in negro children in Brazil and named the disease "ophthalmia brasiliana." In 1883, De Gouvêa [11] of Rio de Janeiro gave the first description of the real picture in xerosis of the conjunctiva, hemeralopia and destruction of the cornea in conjunction with serious cachexia in adult Brazilian slaves. In 1877, Förster [12] reported a case of keratomalacia in a woman, age unknown, suffering from dysentery, which ultimately proved fatal.

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7. Elschnig, in Axenfeld: Lehrbuch der Augenheilkunde, ed. 7, 1923, p. 447.

8. Groenouw: Beziehung der Allgemeinleiden und Organerkrankungen zu Veränderungen und Erkrankungen des Sehorgans, in Graefe-Saemisch: Handbuch der Gesamten Augenheilkunde, ed. 3, Leipzig, Wilhelm Engelmann, 1920.

9. Hildreth: Arch. f. Augenh. u. Ohrenh. **1**:202, 1870.

10. Gama Lobo: Gaseta medica de Lisboa, 1865, p. 430 and 466, quoted by Ullersperger: Brasilianische Augenentzündung, Klin. Monatsbl. f. Augenh. **4**:65, 1866.

11. De Gouvêa: Beiträge zur Kenntnis der Hemeralopie und Xerophthalmie aus Ernährungsstörungen, Graefe's Arch. f. Ophth. **29**:166, 1883.

12. Förster: Keratomalacie bei einen tödlich verlaufendem Falle von Ruhr an beiden Augen einer Patientin, in Graefe-Saemisch: Handbuch der Gesamten Augenheilkunge, ed. 1, Leipzig, Wilhelm Engelmann, 1877, vol. 7, p. 225.

Diseases of the cornea, considered as keratomalacia have also been noted in other serious general diseases. Von Graefe [13] mentioned, for instance, serious cases of cholera in which, toward the end of the stadium algidum, a partial necrosis of the lower portions of the cornea occasionally developed, "a narrow segment of the cornea drying up and turning brown in color. After one or two days the scab falls away, leaving an ulcer surrounded by a grayish area, which may enlarge subsequently." Whether this process is due to keratomalacia, as assumed by Groenouw,[8] rather than to keratitis neuroparalytica is uncertain.

A. Alt [14] reported keratomalacia after typhoid fever and stated that dryness (?) of the conjunctiva during typhoid fever favored the development of diseases of the cornea, especially keratomalacia.

Schmidt-Rimpler [15] described a woman with keratomalacia of both eyes which he considered due to a state of exhaustion after puerperium. Ulcers of the cornea, together with xerosis and night-blindness after scurvy, which developed as a consequence of the failure of the Russian harvest in 1891 and 1892, were observed by Ewmenijew.[16] The ulcers grew rapidly in size, and hypopyon developed which led to perforation of the cornea and frequently to panophthalmitis. It is not possible to infer with any certainty from the brief report whether these conditions occurred in children only or in adults as well.

Hemeralopia with xerosis and keratomalacia was also reported by Bernhard [17] in a drunkard, aged 35, by Schiele [18] in an inmate of a Russian workhouse and by Uhthoff [19] in two undernourished persons in a poorhouse. As far as I can judge from the literature, Flemming's [20] dissertation of 1909 was based on the cases described by Uhthoff. Keratomalacia was reported by Wikerkiewicz [21] in a man, aged 31,

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13. Von Graefe, A.: Ophthalmologische Beobachtungen bei Cholera, Graefe's Arch. f. Ophth. **2**:198, 1866.

14. Alt, A.: Some Remarks on Post-Typhoid Eye Affections, Am. J. Ophth. **9**:347, 1892.

15. Schmidt-Rimpler: Die Erkrankungen des Auges in Zusammenhang mit anderen Erkrankungen, 1898, p. 525.

16. Ewmenijew: Ueber den Einfluss der Miserntен der Jahre 1891-1892 bei der Vevölkerung des Kreises Ostrogostretzk, Westnik Ophth. **13**:473, 1896; abstr., Arch. f. Augenh., 1897, p. 277.

17. Bernhard: Hemeralopie und xerosis conjunctivae bei Potatoren, Diss., Tübingen, 1902.

18. Schiele: Zur Frage der Hemeralopie und Xerosis, Wchnschr. f. Therap. u. Hyg. d. Auges **7**:251, 1904.

19. Uhthoff: Jahresb. d. schlesischen ges. f. vaterl. Kultur und Medizin, 1904, p. 70.

20. Flemming: Zur Keratomalaciefrage unter besonderer Berüchsichtigung des Allgemeinleidens und des Ausganges, Diss., Breslau, 1908.

21. Wikerkiewicz: Einige Bemerkungen die Keratomalacie infolge ungenügender Ernährung betreffend, Post. ocul. 1910; abstr., Michel and Nagel: Jahrsb. u. d. Leisung. im Geb. d. Ophthl. **27**:612, 1910.

who had been a strict vegetarian for ten years. He attributed the disease to insufficient and monotonous food.

Hori [3] and A. Elschnig [22] both mentioned keratomalacia in adults in connection with serious diseases of the liver. In Hori's case, a man, aged 49, with cirrhosis hepatis hypertrophica, chronic inflammation of the intestines and marasmus, corneal ulcers of both eyes developed a few days before death. In Elschnig's case, necrosis of the cornea of both eyes in a woman, aged 54, was found in conjunction with high grade icterus, due to cancer at the mouth of the choledochus.

With the exception of Sauntes [23] who reported keratomalacia in a toothless woman living on mulled beer and rolls soaked in milk, Blegvad [24] was the first in recent years to report on a number of cases of keratomalacia in adults in Europe. In 1917, he found, in an insane asylum, thirteen cases in epidemic form in adults between the ages of 17 and 72. Five patients died, two of bronchopneumonia, two of bronchitis and one of myodegeneratio cordis; of the remainder, one patient became totally blind, two lost the sight of one eye each and one developed leukoma adhaerens. In 1920, he observed another epidemic affecting five inmates of a different insane asylum. The patients' ages ranged between 20 and 50, and in most cases only one eye was affected. In one patient who was reduced to total blindness, the diagnosis was staphyloma corneae. In the same year, Blegvad found an isolated case, that of a man of 59, suffering from xerosis of one eye and keratomalacia of the other. This patient refused further observation.

Apart from these the only other cases reported are by Narog [25] who described two cases of mild keratomalacia in men, aged 18 and 51 respectively, both suffering from chronic intestinal catarrh. Unfortunately, I have only a short abstract of this paper at my disposal.

The incidence of keratomalacia appears to be much greater in the Far East than in the countries of the Western Hemisphere, and xerosis epithelialis appears with still greater frequency, assuming forms and an intensity not found in Europe. I shall deal with this subject at another time. The material furnished in the Far East, however, not only permits one to decide on the principal issue as to whether or not keratomalacia of adults definitely exists, but also enables one to confront the equally important question as to whether keratomalacia has

22. Elschnig, A.: Keratomalacie bei Bindehautxerose, Wien. klin. Wchnschr. 1899, no. 18.

23. Sauntes: Ugesk. f. Laeger. **80**:989, 1918.

24. Blegvad: Xerophthalmia, Keratomalacia and Xerosis Conjunctivae, Am. J. Ophth. **7**:89, 1924.

25. Narog: Beitrag zur Xerosis conjunctivae et corneae, der Hemeralopie, und Keratomalacie, Polska, gaz. leg. **5**:936 and 985, 1926; abstr., Zentralbl. f. d. ges. Ophth. **18**:867, 1927.

the assumed connection with xerosis epithelialis and hemeralopia. If in occidental countries the definite connection has been more or less surely surmised because of the appearance, particularly since the war, of idiopathic xerosis in keratomalacia of children (Blegvad [26] and Birnbacher [27]) and because of the extensive experimental research on the functions of vitamin A in food (P. Knapp,[28] Harden and Zilva,[29] Osborne and Mendel,[30] Freise, Goldschmidt and Frank,[31] Goldschmidt,[32] Hayashi,[33] Yudkin and Lambert [34] and Holm,[35] to single out only a few from the infinite number of papers), in the Far East the connection between xerosis conjunctivae and keratomalacia becomes a certainty, due to nature's experiment, which provides the connecting links. One is thus enabled to increase one's knowledge of a subject which interests more persons than the opththalmologist.

I do not intend to enter into the subject of keratomalacia in childhood in China, although much that is interesting can be found in cases of this sort. In 1927, Hsu of the Peiping Union Medical College reported several clinical cases in the Department of Pediatrics, viewed, however, principally from the standpoint of the pediatrician. He also mentioned the appearance of keratomalacia in patients between 15 and 25 years of age. No particulars regarding these cases were given and no evidence was supplied to show that the disease in question, which was found in poorly nourished, destitute persons, was actually keratomalacia and not one of the other similar diseases of the eye, which are quite unconnected

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26. Blegvad: La xerophthalmie et sa frequence en Danemark de 1909-1920, Compt. rend. Soc. de biol. **89**:197, 1923; Ueber die Xerophthalmie und ihr Vorkommen in Dänemark von 1909-1920, Bibliot. f. Laeger. **115**:300, 1923; footnote 32.

27. Birnbacher: Die epidemische Mangelhemeralopie: Abhandlung aus dem Gebiete der Augenheilkunde, Berlin, S. Karger, 1927, vol. 4.

28. Knapp, P.: Experimenteller Beitrag zur Ernährung von Ratten mit künslicher Nahrung und zum Zusammenhang von Ernährungsstörungen mit Erkrankungen der Konjunktiva, Ztschr. f. exper. Path. u. Therap. **5**:147, 1909.

29. Harden and Zilva: Accessory Factors in the Nutrition of the Rat, Biochem. J. **12**:408, 1918.

30. Osborne, T. B., and Mendel, L. B.: Ophthalmia and Diet, J. A. M. A. **76**:905 (April 2) 1921.

31. Freise, Goldschmidt and Frank: Der Alkoholextrakt aus Vegetabilien als Träger Barlow heilender Stoffe, Monatschr. f. Kinderh. **12**:687, 1913; ibid. **13**:424, 1915.

32. Goldschmidt, M.: Experimenteller Beitrag zur Aetiologie der Keratomalacie, Arch. f. Ophth. **90**:345, 1915.

33. Hayashi: Experimentelle Studien über die Entstehung des Xerophthalmus bei Kaninchen, Tohoku J. Exper. Med. **3**:107, 1922; quoted in Klin. Monatsbl. f. Augenh. **70**:258, 1923.

34. Yudkin and Lambert: Pathogenesis of the Ocular Lesions Produced by a Deficiency of Vitamin, J. Exper. Med. **38**:17, 1923.

35. Holm, E.: Beobachtungen über Xerophthalmie bei Ratten, Graefe's Arch. f. Ophth. **111**:79, 1923.

with poor nourishment and lack of vitamin A in the food. It would appear that some of his older patients had had xerosis and not keratomalacia proper. Verbal information from G. A. M. Hall of Tatungfu (Shansi) and H. H. Shih from Shantung Province mentioned by Hsu [36] in his paper helps to prove that keratomalacia in children is not unusual in other provinces of China. In Peiping, in the Peiping Union Medical College Hospital alone treatment was given in sixty-four cases of infantile keratomalacia in clinics and on the wards from early 1925 to September, 1926.

Elliot [37] in his "Tropical Ophthalmology" spoke only of keratomalacia in children. On the other hand, Wright,[38] in discussing infantile keratomalacia, touched on the subject of keratomalacia in adults, without going into details. It is probable that his statements were based on Kirkpatrick's [39] carefully voiced opinion that "children under the age of 5 are most commonly affected (i.e. by keratomalacia) but it may be seen in older children and even in adults, especially those who are suffering from chronic inflammatory diseases of the intestines." The frequency of keratomalacia in India can be judged from Wright's annual report of 1927, in which he listed 108 cases of keratomalacia in the course of one year in the Government Hospital in Madras. Unfortunately he failed to mention the number of cases of keratomalacia in adults which had come under his personal notice, and only cited them collectively. He contributed, however, various observations of interest on keratomalacia in general, to which I shall refer later. As I do not possess more than an abstract of T. Inouye's paper of 1885, I am not in a position to state whether he spoke of keratomalacia in adults or not. In describing keratomalacia (known as "Kamme") among the children of Japan, he said that it was responsible for 10 per cent of the blindness among Japanese children. My colleague, Grimm, ophthalmologist at the German Hospital in Peiping, told me of a curious corneal disease he had noted in a prison at his former station in the Province of Shantung, which he had taken to be keratomalacia.

The foregoing data represents all the information I have been able to gather on the existence in the Far East generally and in China particularly, of a corneal disease, more or less certainly thought to be keratomalacia.

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36. Hsu, K. L.: Nutritional Keratomalacia: Report of Cases, China M. J. **41**:825, (Oct.) 1927.

37. Elliot, R. H.: Tropical Ophthalmology, London, Oxford University Press, 1920.

38. Wright, R. E.: Keratomalacia in Southern India, Brit. J. Ophth. **6**:164, 1922.

39. Kirkpatrick, quoted by Wright (footnote 36).

The purpose of this report is to demonstrate, with the help of several carefully studied cases, that the process of keratomalacia in adults follows, on the whole, the same course as that in children, and that all conditions from simple xerosis epithelialis—the so-called Bitot's spot—with hemeralopia, by way of xerosis corneae without ulceration of the corneal substance, to the development of the complete picture of corneal necrosis—keratomalacia proper—may also be seen in adults. The second part of this paper deals with the general symptoms observed by me in keratomalacia of adults.

As far as I can judge at the present time, I believe that even though the actual significance of this disease is confined to the East, where the population frequently subsists under unbelievably miserable conditions with a minimum of nourishment, a definite ruling on the existence of keratomalacia in adults is also of importance for Western countries, for guidance in the diagnosis of isolated and uncertain cases of this corneal disease. I remember that during the World War, a man, about 45 years of age, was admitted to the field hospital with a kind of serpiginous ulcer of one cornea. In spite of all kinds of therapy and the greatest care in local treatment, a similar ulcer appeared on the other side several days later, ending in perforation and total blindness. Although a definite xerosis of the conjunctiva could not be found at the time, I do not hesitate today to make a retrospective diagnosis of keratomalacia. The patient had been through all kinds of hardships, including the strain of a long period in the trenches, and in addition was probably suffering from insufficient nourishment, which, by 1917, was general among the people of the Central Powers.

Cases of keratomalacia in adults are also of interest, in my opinion, because of the manifold general symptoms, which are far more easily studied in adults than in children. Children often die of general weakness or of bronchopneumonia before certain symptoms develop, the development of which I have observed repeatedly in adults. On this account, keratomalacia seems to be one symptom of a general "system" disease in the largest sense of the word; that is, it becomes a disease of the entire ectodermal leaf.

Six carefully studied cases are reported in the sequence best suited to pass smoothly from simple but unusually severe xerosis epithelialis conjunctivae, by way of xerosis corneae, to keratomalacia. In the absence of a trained draftsman it has not been possible to provide properly detailed sketches, and I must apologize for the inadequacy of the accompanying drawings, which are intended only to indicate the main points.

REPORT OF CASES

CASE 1.—A laborer, aged 19, was admitted to the hospital on March 26, 1928. He had been sick for four weeks, for the last three of which his vision had been so poor

after sunset that he could find his way about the streets with difficulty. During the preceding four days, the sight of both eyes had decreased rapidly and was much worse than before, even by daylight. He complained of burning and itching of both eyes, particularly the right eye. He had never had any disease of the eyes before, and his vision had always been good. He had been having exceptionally poor nourishment of late, as he could not afford to buy meat more than twice a month; he subsisted on a few vegetables, some white Chinese cabbage and altogether little food, although he was doing heavy manual labor.

Examination.—The patient was pale; his face was puffy and covered with numerous comedones. The skin of the entire body felt dry, as if it had been strewn with coarse powder. The hair was dry and lusterless.

On admission to the hospital, there were various dry rhagades in the outer angles of the lids, covered with dry crusts. The cilia were also covered with a dry secretion, but the margins of the lid were neither reddened nor swollen. There was a hordeolum on the external edge of the left lower lid. The lacrimal glands showed no pathologic condition, but there was decreased lacrimation. The lid conjunctivae were slightly uneven on both sides, with some follicles on the lower lid. There were short rigid folds in both lower fornices; they were grayish yellow like a hyaline degeneration. The upper lid was pale except for a few follicles. The conjunctivae of the eyeballs were dark red, and slightly injected in both eyes,

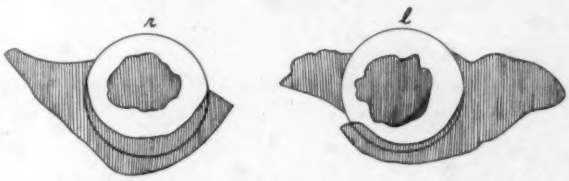

Fig. 1 (case 1).—Extensive xerosis of the conjunctiva of the eyeball in the region of the palpebral fissure and around the upper limbus on the day of admission. The center of the cornea shows a xerosis not connected with the xerosis of the conjunctiva. In this and accompanying illustrations, *r* indicates the right eye, *l* the left eye.

especially in the upper half. There was thickening of the conjunctiva, with the formation of coarse wrinkles, particularly near the limbus. These wrinkles decreased in size the further they were from the limbus. Temporally from the limbus, the conjunctiva was dry and white; typical xerosis extended upward from the palpebral fissure and affected the entire upper limbus, which was covered by the upper lid. In the left eye, the xerosis lay chiefly along the limbus under the upper lid (fig. 1). There were also coarse and faint dry folds in the palpebral fissure.

On the whole, the right cornea was rather dull but even and transparent at the edges. In the center and lying level with the cornea there was a practically circular xerotic opacity, the edge being slightly indented (fig. 1). At its temporal upper edge, however, slight exfoliation of the epithelium could be seen, sharply defined against the clear cornea. The cloudy area was whitish gray, changing to a rich white when the eye was held open for half a minute. There were no blood vessels to be seen, and no symptoms of irritation were present. The xerosis of the conjunctiva continued under cover of the upper lid to the corneal limbus, forming a sickle-shaped, pannus-like area through which the normal limbus vessels were visible.

The left cornea was slightly dull. In the center there was a faint white opacity (fig. 1), dry, with definite stippling. No blood vessels were visible. There was the same pannus-like covering of the limbus by a xerotic zone as in the right cornea. The anterior chamber and iris were normal. The shagreen of the lens had definitely lost in power of reflection and showed grayish-brown and greenish coloring. The entire left lens seemed slightly dim, but only the periphery could be examined in the right. In the direct light the lens reflected rather more than usual and there were numerous subcapsular spot-shaped opacities. The fundus was normal on both sides.

Vision was 6/30 in both eyes, according to the central xerosis. Sensibility of the cornea was definitely diminished on both sides.

Scrapings from the xerosis of the cornea and conjunctiva showed numerous xerosis bacilli and a few pneumococci on the degenerated epithelial cells.

Course of Disease.—March 27, 1928: The xerosis of the eyeball conjunctiva in the right eye had broken up into several large-sized islands. The dimness of the

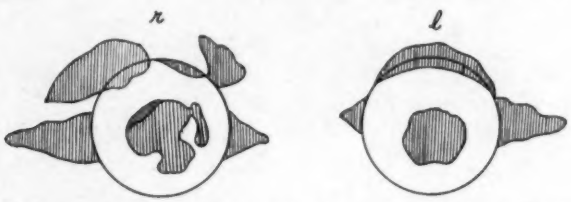

Fig. 2 (case 1).—One day after admission: The xerosis of the conjunctiva of the eyeball and cornea becomes smaller.

Fig. 3 (case 1).—Four days after admission: The xerosis of the conjunctiva of the right eyeball and cornea is broken up into different irregular islands. The xerosis of the left eye has almost disappeared. The left cornea shows only a faint haziness following the former xerosis.

cornea had disappeared, but xerosis of the limbus was still present (fig. 2). The borders of the corneal xerosis were indefinite, narrowing toward the lower half. A small island had almost isolated itself nasally. The folds of the conjunctiva of the eyeball continued unchanged. The xerosis of the limbus in the left eye was clearly seen (fig. 2). The corneal opacity was less sharp and rather smaller in size; the surface stippling was still evident; the marginal parts of the cornea were clear. It was still possible, by holding open the lids for about one minute, not only to intensify the opacity in the center, but also to cause blurring of the unclouded part of the cornea. There was practically no lacrimation.

March 29: The xerotic islands of the conjunctiva in the right eye had become smaller; the folds were practically unchanged. The corneal xerosis was divided into numerous small and irregular islands (fig. 3). The xerosis of the conjunc-

tiva in the left eye had disappeared with the exception of a Bitot's spot in the temporal palpebral fissure and one small island. No actual xerosis of the corneal center remained, but in its place was a filmy disk-shaped area (fig. 3).

March 31: In the right eye there were folds of the moderately injected bulbar conjunctiva on the nasal side of the cornea. Xerosis had disappeared, with the exception of a small triangular spot temporally. The xerotic islands of the cornea, four in number and very faint, had moved further apart (fig. 4). The sickle-shaped spot where the exfoliation of the epithelium had taken place, remained the only opacity of importance. The marginal parts of the cornea had cleared up and all xerosis of the limbus had disappeared.

In the left eye temporally from the limbus, the remains of the bulbar xerosis were seen. Slightly below the center of the cornea, the filmlike opacity was just recognizable (fig. 4).

Fig. 4 (case 1).—Six days after admission: The various xerotic islands of the right cornea have become smaller in size; the xerosis of the conjunctiva is almost gone in both eyes. The left cornea shows only a faint haziness.

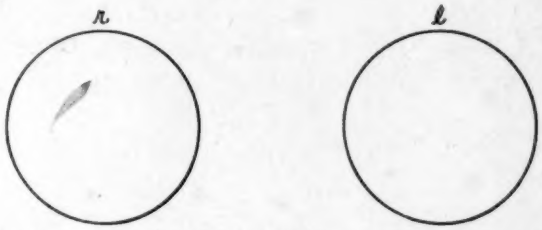

Fig. 5 (case 1).—Eleven days after admission: The xerosis of the conjunctiva has entirely disappeared; the right cornea shows an epithelial defect remaining from the corneal xerosis.

The slit-lamp showed the same changes of the shagreen of the lens in the right eye as in the left, where they were still visible.

Examination of the blood showed 4,600,000 red blood cells, 7,000 white blood cells, polymorphonuclears, 80 per cent; lymphocytes, 20 per cent, and hemoglobin, 75 per cent. The urine was slightly alkaline with a specific gravity of 1.024; the stools were normal. The temperature rose (fig. 6) to 38 C. (100.4 F.). The chemical examination of the blood showed 5 mg. of phosphates and 10 mg. of calcium. The Wassermann reaction was negative. The medical examination showed poor general nutrition, normal heart and lungs and the suggestion of a mild tetany.

April 5: Condition on discharge: In the right eye, the conjunctiva of the lids was pale; there were still some folds in the lower fornices. The folds in the bulbar conjunctiva had not altogether disappeared but xerosis was entirely absent.

On the temporal side of the center of the right cornea it was still possible to distinguish a faint sickle-shaped dimness (fig. 5), which was the last of the xerosis with its loss of epithelium. Except for this, the cornea was bright and normal. The left eye was entirely normal. The shagreen of the lens was still slightly dull on both sides.

Comment.—A laboring man, aged 19, when admitted to the hospital, had ashen skin and was poorly nourished; his entire face was covered with comedones. There were rhagades in the outer angles of the lids. After about three weeks of night blindness a wrinkling of the bulbar conjunctiva in the palpebral fissure appeared, and there was under the protecting upper lid an atypical xerosis, which covered the margins of the cornea adjoining the limbus like a sickle-shaped pannus. Xerosis was also present in the center of the cornea. Between the affected corneal margin and the xerotic center there was an area permanently free from xerosis, though slightly opaque in the beginning. Under vitamin treatment the disk-shaped xerosis of the corneal center

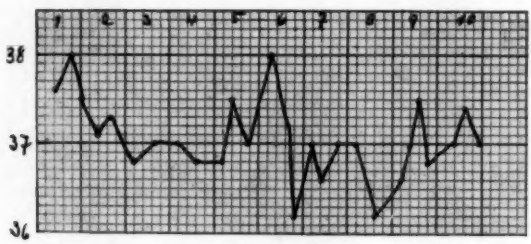

Fig. 6 (case 1).—Temperature curve.

disappeared within five days in the left eye and within eleven days in the right, the affected area breaking up into separate islands, which changed both their position and their relation to each other in the process of healing. During the period of observation there was only one place in the right eye in which just recognizable desquamation of the epithelium was visible to the naked eye. This resulted in a small defect of the epithelium. The bulbar xerosis was also disappearing by breaking up into several islands. The symptoms which lasted the longest were a faint wrinkle formation of the conjunctiva in the palpebral fissure region and a barely perceptible lack of luster of the conjunctiva above the limbus. Other local symptoms of the eye which should be mentioned were night blindness and a curious decrease in the luster of the anterior surface of the lens, which showed itself in a decrease of brightness of the anterior lens shagreen under slit-lamp illumination. Grayish-brown tones predominated in the shagreen picture. In view of the difficulties of examination, owing to the dulness of the corneal center, nothing positive can be asserted regarding changes of the fundus, but there were no signs of marked importance.

Among the general symptoms, there was a slight rise of temperature to 38 C. (100.4 F.), which lasted until the sixth day of observation, the day on which the left eye recovered. The department of medicine emphasized the fact that no cause for the rise in temperature could be found. The comedones on the facial epidermis which probably indicated a cessation of the functions of the sebaceous glands, the dry and ashy skin, the pasty, puffy face, the lifeless hair and all the other general symptoms noted on admission had either become less or completely disappeared at the time of discharge. Although the red blood count was normal and the distribution of the white cells practically normal, the blood showed a slight oligochromemia (75 per cent hemoglobin). The blood count for this patient unfortunately was not repeated at the time of discharge.

No local treatment was given, but a diet of butter, milk, liver, cod liver oil and lemon juice was prescribed. As a result of this diet, the condition of the eye and the general symptoms entirely disappeared.

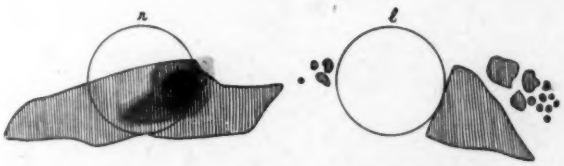

Fig. 7 (case 2).—On day of admission: Extensive xerosis is present in the right cornea and conjunctiva of the eyeball; dense old scars in the nasal lower sector of the cornea, and irregular Bitot's spots on the conjunctiva of the left eyeball.

Case 2.—A student, aged 19, was admitted to the hospital on July 9, 1928. He stated that for ten years his right eye had been weaker than the left and that during the last forty days, vision in the right eye had still further decreased. Neither he nor his father was able to give further details of the illness of ten years before.

Examination.—The conjunctiva of the lids had a normal luster and was slightly hyperemic. In spite of the extent of the disease in the cornea the right bulbus was pale, only a few large vessels being visible. The conjunctiva of the lower half of the bulbus of each eye was, on the other hand, light brown and had lost its luster, more so in the right eye than in the left. There were typical but unusually large Bitot's spots in the region of the palpebral fissure (fig. 7), from which folds extended in various directions, temporally more than nasally. The depressions of the folds were free from xerosis, but not the elevated parts. Approaching the temporal angle of the lid and the caruncle, the folds became small isolated wrinkles until finally there was an area of normal conjunctiva lacking only in normal luster. The xerotic zone surrounded the lower limbus in the right eye (fig. 7).

In the left eye there were a few spots on the nasal side so faint as to be easily overlooked, but temporally a sharp xerotic triangle approached the cornea at a tangent. In addition there were a few minute, separate spots on the upper temporal side of this typical Bitot's spot (fig. 7).

In the right cornea an old flat scar in the nasal lower quadrant showed a loss of epithelium in its densest part, a yellowish discoloration and extreme dryness. The xerotic zone covered the entire lower half of the cornea and was so directly connected with the limbus, the part most affected by the xerosis, that the normal boundary between cornea and sclera was hardly distinguishable. Some of the more dense xerotic areas were slightly indented by desquamation of the epithelial cells. The upper boundary of the xerosis was clearly defined by a horizontal line consisting of tiny isolated white xerotic spots. Besides the limbus, the region of the scar was the part most affected by the xerosis. In the upper half of the cornea where the luster was practically normal, only a few faintly gray xerotic islands could be seen with the help of a strong focal illumination.

The left cornea was normal. The sensibility of the right cornea was almost nil; that of the left was slightly diminished. The anterior chamber, iris and lens showed no evidence of disease.

Slit-lamp examination showed a faint protruding network of limbus capillaries. When magnified twenty times, the entire center of the cornea was seen to be strewn irregularly with many white xerotic spots, separated from each other. Some glistening spots represented reflexes of small concave epithelium hollows. The indirect light gave a picture of superficial bedewing, through which the

Fig. 8 (case 2).—Four days after admission: Xerosis of the cornea and conjunctiva has decreased and is breaking up into different islands. The corneal scars are omitted in this and the following sketch.

xerotic spots were distributed. The corneal posterior surface was normal. Even the reflex pictures showed no change of the endothelium. The shagreen of the lens was somewhat duller than normal, and there were numerous white spotlike opacities of both cortices.

As far as the patient could be made to understand the questions, his eyesight apparently was rather worse after sundown. (Unfortunately, no photometer was at hand for measuring the adaptation at that time.)

Course of Disease.—July 13, 1928: The xerotic spots in the right eye were smaller, lying half on the conjunctiva and half on the cornea (fig. 8). The upper horizontal boundary had disappeared. On the scar a dense xerotic island was at no point connected with the marginal xerosis of the cornea. Two sickle-shaped patches of marked dryness adjoined the lower limbus. There was one spherical xerotic island on the nasal side, and on the temporal side of the limbus the wrinkle formation was still distinct, but the rest of the conjunctiva was practically normal. The xerosis in the conjunctiva of the left eye was smaller than on admission and now appeared in the shape of two islands (fig. 8).

July 14: A piece of the yellowish conjunctiva was excised from the lower fornix of the right eye.

July 18: The conjunctiva of the lid, especially in the fornix, was still rough, grayish yellow, wrinkled, and worse in the right eye than in the left.

The xerotic area on the temporal side of the right cornea had decreased in size (fig. 9) and was sharply defined; an intense yellow discoloration near the limbus on the cornea, between 3 and 5 o'clock, looked like a superficial deposits. On the nasal side of the cornea there was a small elongated island, also slightly yellow, at the margin of the corneal scar. The central xerosis which had covered the densest part of the scar, had vanished, but there were a quantity of minute, shiny hollows lying nearer the center of the cornea, after the manner of a herpes corneae in the early stages.

Near the limbus of the left eye there was one xerotic spot on the temporal side, which had already become bright in the center, and showed a large quantity of tiny irregularities where the xerotic scabs had been exfoliated (fig. 9).

July 26: There was still some dryness in the opacity of the lower half of the right cornea but the xerosis had disappeared, as it had also in the bulbar conjunctiva.

Medical examination showed: lymph glands at the neck; normal heart and lungs and the liver and spleen not palpable. The liver function test with tetrachlorphthalein gave normal results. The tonsils were injected, but otherwise the nose and throat were normal.

Examination of the blood on July 11, showed 4,800,000 red blood cells, 9,200 white blood cells; lymphocytes, 78 per cent; leukocytes, 20 per cent, and hemo-

Fig. 9 (case 2).—Nine days after admission: The xerosis has become smaller and has changed its position to a certain extent. Many small hollows are seen in the center of the right cornea and in the Bitot's spot of the left eye.

globin, 85 per cent. On July 28, there were 5,400,000 red blood cells, 9,200 white blood cells; lymphocytes, 61 per cent; leukocytes, 31 per cent, and hemoglobin, 75 per cent. The urine was alkaline, with specific gravity of 1.015. Many phosphates and epithelial cells were present. The Wassermann reaction was negative. The stool proved normal in three examinations. The temperature was generally over 37 C. (98.6 F.) and for two days it was 38 C. (100.4 F.) (fig. 10).

Histologic Data.—The excised section was a part of the lower fornix of the right eye and was removed, after cocainization, by Wecker's scissors, fixed in sublimate-glacial acetic acid, and stained according to the various methods.

The epithelium had from eight to ten layers, was continuous, and showed a thick layer of keratohyaline, many cells of which were in process of exfoliation. The five or six layers in the middle consisted of cubic cells; the position of the basal cells was normal, and the posterior borderline of the epithelium was wavy with small pointed cones and indentations at the back. The presence of a number of epithelial hollows (Elschnig) which might be situated in any one of the layers, with a diameter of from two to six cells was of interest. A few exfoliated epithelial cells and some granular pigment lay in the center of these hollows.

What was most striking was the uninterrupted pigment line in the basal cell layer, which could be seen with low magnification. By examination with the immersion lens the uniform layer dissolved itself into lines of light-brown pigment

which lay between the basal cells, continuing on through three or four cell layers toward the surface. There was also a certain amount of pigment inside the basal cells themselves. It was remarkable how plentiful was the pigmentation of practically all the epithelial cells to the topmost layer. This pigment was granular and dark brown, surmounting the nuclei of the epithelial cells like a cap and was found exclusively at the pole of the nucleus that was directed toward the surface.

The mucosa showed streaklike lymphocytic infiltrations, which were situated chiefly along the smaller superficial vessels. The larger vessels were practically free from changes. The connective tissue was also slightly infiltrated on the surface side with occasional plasma cells.

In summarizing the histologic observations on the conjunctiva of the lower fornix, the fact that the pavement epithelium assumed the appearance of the external skin and that there were both hyperkeratosis and parakeratosis was of importance. At the same time that dense pigmentation of the basal cell layer set in, similar to the pigmentation of the exterior skin, there was only a slight lymphocytic infiltration of the superficial parts of the mucosa.

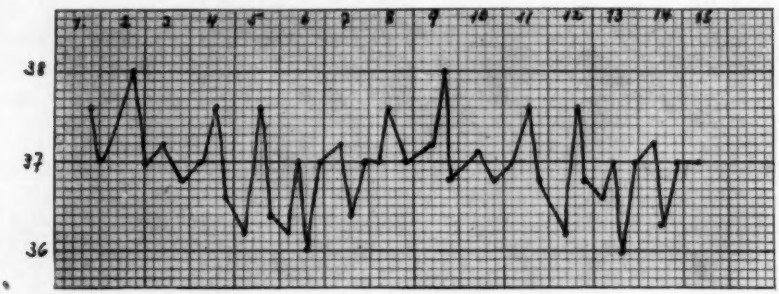

Fig. 10 (case 2).—Temperature curve.

Comment.—This youth of 19, poorly nourished and of indifferent intellect, represented the type in which the xerosis of the eyeball conjunctiva in the right eye (the left eye was in much better condition) continued into the cornea and finally covered the entire lower half with a dense xerotic layer. The xerosis of the conjunctiva occupied practically the entire lower half of the bulbus and was furrowed by coarse folds. The lower fornix conjunctiva, only slightly injected, showed deep but short horizontal folds, the shape of which did not change even when the conjunctiva was stretched. The discoloration here and in the lower half of the bulbus was yellow and yellowish brown. The corneal xerosis left off abruptly at the top, its densest part being on the site of an old corneal scar. At this point, the surface layers of the epithelium had crumbled and a pale yellow exfoliated area had formed. Receding in the direction of the limbus, the xerosis first broke up into islands, causing tiny superficial defects of the epithelium of the cornea at various points. Owing to the patient's refusal to take the prescribed diet, there were several minor relapses, and recovery was delayed. As in case

1, there was a mild, though slightly uncertain night blindness and the same dulness of the shagreen of the anterior lens with similar brown tones.

General symptoms in this case were confined to the typical earthy texture of the skin, as if it were covered with flour, numerous comedones of the face, moderate diarrhea during the first period in hospital, slight swelling of the lymph glands of the neck and dry, dull, lifeless hair. The mucous membranes of the mouth were slightly hyperemic, the tonsils were enlarged, and the liver and spleen not palpable. The liver function test with phenoltetrachlorphthalein gave normal results. There were white blood cells and many epithelial cells in the urine, which was alkaline and otherwise normal.

Although the count of the red blood cells was normal and that of the white blood cells practically so, the hemoglobin showed a decrease from 85 per cent at the first test to 75 per cent at the second. The Wassermann reaction and stool cultures were negative. The temperature varied between 37 and 38 C.

CASE 3.—A student, aged 26, was admitted to the hospital on July 13, 1928. He had had inflammation of the eyes for three months, during which time he had been treated in a provincial hospital without obtaining relief. For several weeks he had had diarrhea, with five or six movements a day. At the age of 11, he had had frequent inflammations of the eye.

Examination.—The skin was ashen and dry and felt as if it had been powdered. There were numerous comedones on the face. The hair was dry.

On admission to the hospital the patient showed a slight photophobia, and the lids were puffy and swollen. No signs of inflammation were apparent.

The conjunctivae of both lower lids were mildly injected and grayish yellow. No scars were visible. In the lower fornix, which was otherwise normal and not inflamed, there were several grayish-yellow horizontal rolls with the appearance of follicles joined together as in trachoma or as in the first stages of a hyaline degeneration. The conjunctiva was much drier than normal. There was a discharge of long threads from a fibrinous secretion which adhered firmly to the palpebral conjunctiva.

The bulbar conjunctiva was gray with a yellowish tinge and showed little congestion. Bitot's spots were seen in the typical places (fig. 11). In addition the upper limbus of both eyes was covered, in the form of a half-moon, with a dense xerosis, which was also apparent on the corneal scars. In the right eye, in 4 and in 9 o'clock, there were phlyctenulae.

In the center and periphery of the right cornea there were many old maculae corneae of varying density, probably the result of previous keratitis eczematosa. In the right eye the maculae adjoined the limbus. In the upper quarter the epithelium of the cornea, connected with the sickle-shaped zone of the conjunctiva, was xerotic and reached almost to the center. The xerosis was not equally dense all over, in parts seeming to have fallen off. The lower edge was sharply outlined. In addition to the discarded xerotic masses there were minute superficial exfoliated areas which did not, however, assume fluorescein coloring. In the center of the left cornea there were several small maculae corneae (fig. 11). In continuation of the limbus the upper part of the cornea was covered by a sickle-

shaped xerotic layer, which was quite independent of the borders of the maculae. There were no recent inflammatory symptoms in either of the corneas. Some old sclerotic vessels were present.

Slit-lamp examination revealed, in addition to many epithelial cells which were in process of exfoliation, a remarkable number of vacuoles in the epithelial layer, but no crystals (Narog[22]). The posterior surface of the cornea showed several particles of old pigment. The endothelium was normal even in the examination with reflex zones. There was marked dulness of the shagreen of the lens in the left eye, clearly visible with the slit-lamp. Several dotlike opacities were seen at the anterior and posterior cortex. It was not possible to judge of the posterior shagreen as it was not clearly visible.

Examination of the fundis revealed an old central and peripheral chorioretinitis proliferans interna in the left eye.

Fig. 11 (case 3).—On the day of admission: The xerosis is lying especially on the upper limbus and upper half of the cornea, covering in part the numerous old corneal scars; irregular xerosis of the conjunctiva of the eyeball is present.

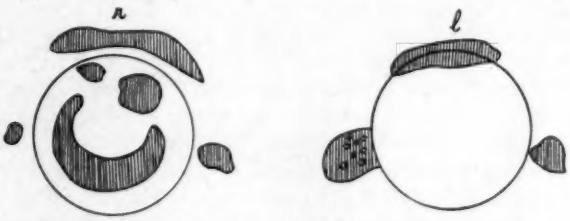

Fig. 12 (case 3).—Five days after admission: The corneal xerosis of the right eye has entirely changed its position and is broken up into various islands. (The corneal scars are omitted in this and the following sketch.) Some epithelial hollows are present in the nasal Bitot's spot of the left eye.

Vision in the right eye was reduced to counting of fingers at a distance of half a meter. In the left eye, vision was 5/60, owing to the lesions of the fundus.

Course of Disease.—July 14, 1928: Xerosis of the right eye had largely disappeared in the upper half, but a sickle-shaped xerotic patch of the cornea with sharply defined edges had suddenly appeared over night on the border of the old corneal opacities (fig. 12). There were only two small xerotic spots at the top. The cornea was on the whole improved in luster, compared with the days before. There were a few shining areas of exfoliated xerotic plaques.

The left cornea was free from xerosis. On the temporal side of the cornea the Bitot's spot was still present. On the nasal side the conjunctiva was shining but slightly wrinkled. The upper part of the limbus was still rather dry, but there was no actual xerosis (fig. 12).

The patient had had six stools the day before, against two or three on the preceding days. He had no appetite, and he refused liver and cod liver oil.

July 18: The number of comedones in the face had decreased remarkably, remaining on the forehead and temple only. The skin felt greasy.

The conjunctiva of the lids was still dry and slightly wrinkled. The upper lids were normal, but the lower fornix was still very uneven.

The xerosis of the right cornea had again completely changed its position, being densest in the nasal upper quadrant on the densest part of the old corneal scar (fig. 13). The upper limbus was free and the xerotic half-moon on the temporal side of the corneal scar had entirely disappeared. On the nasal side of the limbus there was a fairly large area of numerous small hollows.

Fig. 13 (case 3).—Nine days after admission: The corneal xerosis has again changed its shape and position. Epithelial hollows are present in the conjunctiva of the right eyeball (nasally). The left eye shows no more xerosis.

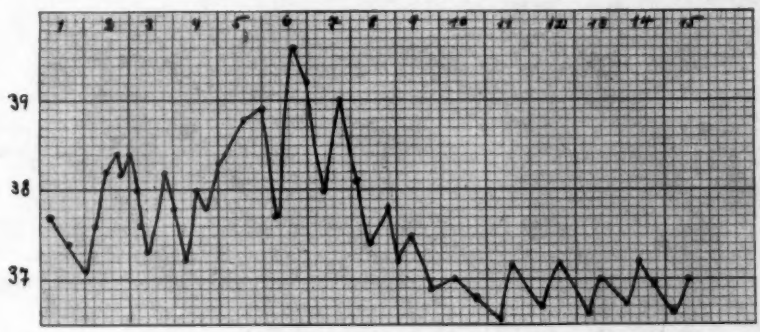

Fig. 14 (case 3).—Temperature curve.

The cornea and conjunctiva of the left eye were normal.

Examination of the blood showed: 4,600,000 red blood cells; 10,000 white blood cells; polymorphonuclears, 82 per cent; lymphocytes, 28 per cent, and hemoglobin 70 per cent. A fortnight later there were 4,500,000 red cells, 10,000 white cells, and the polymorphonuclears, lymphocytes and hemoglobin remained unchanged. The temperature varied between 37 and 39.6 C. (between 98.6 and 103.2 F.) during the first six days (fig. 14). Three examinations showed a normal stool, except for the presence of *Ascaris*. Special and repeated examination for dysentery also gave negative results. The Wassermann reaction was negative. The urine had a specific gravity of 1.018; there was no albumin or sugar. The sediment contained many white cells and triple phosphates, but no epithelial cells. Tests for melanin and for bile were negative. The tonsils and pharynx were slightly discolored and

rather dry. Medical examination showed no pathologic condition. The liver and spleen were normal. The liver function test with phenoltetrachlorphthalein gave normal results. No cause for a rise in temperature could be found medically.

July 25: On discharge, the xerosis of the conjunctiva and cornea had entirely disappeared on both sides. The conjunctiva of the lid again showed a normal luster; only the conjunctiva of the fornices and of the bulbus was yellow. Vision in the right eye was 6/30 and in the left, 5/60. In spite of general improvement, the hemoglobin content was only 70 per cent.

Comment.—In this student, aged 26, the disease of the eye was preceded by an ordinary diarrhea of three weeks' duration; during his stay in the hospital no dysentery or other infectious intestinal disease was discovered. The chief characteristic of this case was the fact that as in case 1 the corneal xerosis developed under the upper lid and proceeded in a continuous line toward the cornea. It cannot be denied that xerosis has a predilection for the site of old scars. The interesting point in this case was the tendency of the xerosis to jump from one point to another, moving into areas as yet unaffected. These changes usually took place within a period of twenty-four hours. Whether these changes from bad to worse and vice versa can be explained by the relapses of the diarrhea is uncertain, but the first notable change of position of the xerosis was preceded by an increase in loose bowel movements. Under vitamin treatment the condition of the eyes was completely cured in a fortnight. The lens was affected in this case also, the shagreen of the lens being dull and oscillating slightly in various colors. It should be emphasized that the endothelium of the cornea remained normal on repeated examination, which was also true in all three of the cases described so far.

Among general symptoms should be mentioned the ashy-grayness and earthy consistency of the skin, the dulness of the hair and the numerous comedones on the face, which had practically vanished at the time of discharge. The urine contained many phosphates, and white blood cells, but practically no epithelial cells. The tonsils were inflamed, and the mucous membranes of the mouth and throat showed a livid discoloration and were rather dry. Results of the medical examination were entirely negative. The liver function was normal. In spite of a normal blood count, the hemoglobin content was only 70 per cent, thus indicating another case of oligochromemia. It should be noted that the diarrhea ceased abruptly as soon as the rather sudden turn for the better set in.

Case 4.—A weaver, aged 18, was admitted to the hospital on March 21, 1928. The patient had complained of pain and inflammation of the eyes for two weeks. During this time, he had found his vision extremely poor in the evening, especially at dusk. He had been suffering from severe pains in the left eye for two days, and vision had decreased at the same time, even during the day. His vision had always been good, and he did not remember ever having suffered from a disease of the eyes before. For months his diet had been rice and peas.

On physical examination, the patient was found to be very anemic and undernourished. He was pale, and there were many comedones over the entire face. Both his skin and hair were dry.

Examination of the Eye.—On admission, the patient showed, in the conjunctiva of the right lid, especially in the upper lid, mild papillary hypertrophia with a few follicles which might indicate trachoma. Near the margin of the upper conjunctiva there were two small flat ulcers almost joining, with sharply defined margins; to all appearance, they were phlyctenulae of the conjunctiva of the lid. In the center of the upper lid there was a minute gray spot, a beginning phlyctenula. The conjunctiva of the lower lid was practically normal. The conjunctiva of the left lid showed also mild papillary hypertrophy with a few follicles. The dark gray discoloration of the lower fornix was striking, especially on the left, and might easily be mistaken for argyrosis. This discoloration was also found in the conjunctiva of the eyeball. In the right eye, it was pale yellow, while in the left it mingled with the injection, which was probably the reason for its peculiarly dull appearance. The lower fornix showed stiff unyielding folds, like a hyaline degeneration of the conjunctiva. Two typical Bitot's spots were noticed on the right bulbar conjunctiva. The conjunctiva surrounding the cornea of the left eye was much wrinkled and dry. The injection of the blood vessels which was evenly distributed gave it a dull red appearance (fig. 15). The conjunctiva of the eyeball, rather swollen on the whole, was separated by a step from the cornea. The xerosis appeared in a few places only, in the form of white isolated plaques. The conjunctiva was rather dry, like the exterior skin, with few characteristics of mucous membrane remaining.

The right cornea was slightly and homogeneously dull, although no further changes were observed. The whole left cornea was dull and dry, but translucent at the margins. At a distance of 1.5 mm. from the limbus there was a dense white ring-shaped opacity. About in the middle of the cornea, somewhat on the lower nasal side, there was a circular ulcer, a kind of ulcus serpens, with undermined white edges and base (fig. 15). The edges were not clearly defined and were somewhat jagged, without vessels. Lower down behind the ulcer the cornea showed an abscess in the depth to which a hypopyon 4 mm. high was joined. The center of the ulcer was slightly raised above the level of the cornea which indicated its thinness. Its size was 5 by 5 mm.

Sensibility of the cornea was decreased on both sides. The left anterior chamber was present, and shallow, though dull, as a result of pus. The right iris was normal. The left iris as much as was visible was also normal. The left pupil was somewhat smaller than the right and was circular. The tonus of the left eye was lower than that of the right.

Bacteriologic examination of the ulcer of the left eye showed, on dead epithelial cells, numerous xerosis bacilli and pneumococci, fewer but still numerous diplobacilli, and diplococci similar to staphylococci or sarcinae. Many of these germs grew turflike on the degenerating epithelial cells and were also found in quantities on the mucus. The scraping taken from the left bulbar conjunctiva showed practically the same picture as on the cornea, four kinds of germs being seen with a predominance of the xerosis bacillus.

Numerous xerosis bacilli were found on the hazy cornea of the right eye.

The vision of the right eye was 6/15, and that of the left was hand movement before the eye. Light sense and projection of the left eye were good.

Course of Disease.—March 22, 1928: The bulbar conjunctiva of the left eye was unchanged. The edges of the ulcer of the left cornea were more sharply defined, so that the corneal process stood in greater contrast to the hypopyon. The

corneal margins had cleared considerably. No local treatment was given, but directly after admission a diet of cod liver oil, liver, fresh fruit, vegetables and butter was prescribed. Bacteriologic examination showed the same germs as the day before, in about the same quantities.

March 24: The size of the ulcer in the left eye was 4 by 4 mm. The edges were sharply defined. There were symptoms of clearing up and further thinning of the bottom of the ulcer.

March 29: The conjunctiva of the left eye showed no more xerosis except in the temporal lower quadrant. The corneal ulcer was smaller, its edges sharply defined, with a few superficial vessels growing toward the lower margin. The base of the ulcer was more elevated. The marginal parts of the cornea were still edematous and opaque. The anterior chamber was deep; the hypopyon had disappeared. As far as the iris could be seen, it was almost normal.

March 31: The conjunctiva of the lid in both eyes was only slightly uneven, and was otherwise normal; the previous diagnosis of trachoma, therefore, seemed improbable.

The right bulbar conjunctiva was normal in pallor, except for a small triangular Bitot's spot on the temporal side of the limbus. There was no injection and the cornea had its normal luster.

The left eyeball was slightly injected all around the cornea. The conjunctiva was dry. The xerosis, however, had totally disappeared. The ulcer of the left cornea had greatly decreased in size. The surrounding cornea was clear, but the peripheral zones showed an arcus senilis-shaped dense ring opacity. The anterior chamber was of normal depth and its contents were normal. The iris was clearly visible, without pathologic changes. Bacteriologic examination showed the left cornea practically free from germs, only a few xerosis bacilli remaining.

The urine was straw colored and clear, with a specific gravity of 1.014 and hardly any sediment. Examination of the blood showed: 4,300,000 red blood cells, 5,800 white blood cells; hemoglobin, 80 per cent; polymorphonuclears, 64 per cent; lymphocytes, 28 per cent; mononuclears, 7 per cent, and eosinophils, 1 per cent. The stool was normal except for the presence of *Ascaris*. The Wassermann reaction was negative. The maximum temperature was 37.4 C. (99.3 F.).

April 15: The general condition had improved considerably. The patient looked healthier, his skin was shiny and damp and the comedones had disappeared. The lower fornix of both eyes was uneven while the upper fornix remained perfectly normal. The conjunctiva of the right eyeball was shiny and its color normal; the cornea was clear and bright. No xerosis was present in the conjunctiva of the left eyeball but there was a slight pericorneal injection at the lower margin. The entire conjunctiva was still thicker than normal. The corneal ulcer was healed and had become a dense white opacity surrounded by a gray area. Extending inward from the somewhat enlarged limbus there was a broad gray belt, which appeared to be separated from the limbus by transparent corneal tissue; a "gerontoxon" (fatty degeneration) which had developed in the course of the disease. In the lower half of the corneal posterior surface some deposits of fibrin and pigment could be seen. The anterior chamber was of normal depth. In the left iris there was a posterior synechia, from 6 to 9 o'clock, visible only after the use of atropine. The iris stroma was otherwise normal. Vision in the right eye was 6/10; that in the left was limited to the counting of fingers at a distance of 5 meters.

Comment.—This man, aged 18, undernourished, with ashen-gray complexion, gave the picture usually found in adults with keratomalacia:

On superficial examination the circular ulcer which is often not exactly in the center, gave the impression of an ulcus serpens with dense white infiltration, the margin slightly raised but generally standing in sharp contrast to the rest of the cornea. A second infiltration, lying in the deeper layers of the cornea, suggested a posterior abscess, such as is also generally found in the early stages of serpent ulcers. In such cases the hypopyon, seldom fluid, completes the false impression of an ulcus serpens. The whole ulcer was devoid of all vessels at the beginning. In this case a typical xerosis of the rest of the cornea was lacking, although the marginal parts were of a decided smoky-grayish dulness and even more dim than is apparent in figure 15.

The examination of the conjunctiva is the only guard against a mistaken diagnosis. In the lower fornix of this patient were also found acute, dirty-gray folds, with the appearance of severe argyrosis. The entire bulbar conjunctiva had become a dense leathery, dried-up membrane lying in circular folds, such as are otherwise never found in inflammatory conditions of the eye. These circular folds surrounded the cornea like a dam, and on the ridges were found numerous white xerotic deposits, although the hollows were free from these. (The xerosis was omitted in figure 15 in order to emphasize better the color of the conjunctiva.) The peculiar dull mauve-red color of the bulbar conjunctiva, which at times showed a yellow tinge, was particularly characteristic, and could hardly have been mistaken for the reddish hue of an inflammatory process. In spite of its advanced condition the ulcer healed completely within ten days as a result of proper diet only, without any local treatment. To the eye itself atropine was applied, in order to safeguard the iris from the threatened area in case of perforation. The purulent fibrinous exudate in the anterior chamber disappeared, leaving behind fine precipitates on the posterior surface of the cornea and a few spots of brown pigment on the anterior capsule of the lens as well as a synechia of brown pigment which later on was expanded by atrophine until it tore.

The folds of the conjunctiva of the bulbus and lower fornix remained long after the disappearance of the xerosis which in this case had not been severe. The right eye which had been less seriously affected lost the Bitot's spots within a week.

This patient also had the characteristic gray color of the skin, and his face was somewhat puffy, covered with numberless comedones. The skin of the body was dry as if powdered with flour, with countless small wrinkles, and with a tendency toward furuncles. The hair was dry and dull.

The stool and urine were normal and the Wassermann reaction was negative. There was a slight decrease of red blood cells and a proportionate lowering of the hemoglobin to 80 per cent. The blood also

showed relative lymphocytosis and 7 per cent transitional forms. The temperature was subfebrile.

I should mention here the formation of a gerontoxon-like opacity in the left eye during the period of clinical observation, which has not disappeared up to the present time, seven months later. The limbus was first covered with the folds of the leathery bulbar conjunctiva, as well as by marginal xerosis of the cornea, so that no details could be distinguished. After the decrease in the xerosis, a whitish-gray, indistinctly defined circle was formed concentric with the limbus (at first, 2 mm. in width). In the region of the lower chamber angle this could not be clearly distinguished from the deep corneal infiltration and the hypopyon in the anterior chamber. During the decrease of the pathologic symptoms in the cornea and anterior chamber this newly formed ring withdrew increasingly toward the limbus, becoming whiter, narrower and more sharply defined. From this time on, it was separated from the clearly visible limbus by a normal unclouded zone and resembled a genuine gerontoxon. During the entire period of observation the opacity remained under the epithelium and gave the impression of being deep-seated in the parenchyma, but the narrow beam of the slit-lamp showed that it did not extend farther than the middle of the parenchyma, which after further enlargement (objective a2, ocular 4) proved to be formed of minute regular dots and flakes of equal size. In this respect, the opacity was also like a gerontoxon. The network of capillaries of the corneal limbus stopped just before the gerontoxon. The zone in front of Descemet's membrane was clear. It should be emphasized that there was no trace of arcus senilis in the other eye, so that the formation in the left eye is rightly thought to be connected with the corneal process of keratomalacia.

My impression of the origin of this gerontoxon is as follows: the broad white circle which was seen in the beginning of the keratomalacia was a kind of wall of demarcation between the severely altered, necrotic corneal center and the periphery, which, because of the nearness of the limbus capillaries, was still comparatively well-nourished. This circle, probably consisting of leukocytes, which lay under the Bowman's membrane, deteriorated into a kind of fatty degeneration and remained in this condition. This is easily understood in the case of the cornea, where the metabolism was extensively changed through the severity of the disease. Only prolonged observation can tell whether this is a permanent formation or merely a temporary one, but in view of the foregoing facts, I am inclined to believe it permanent. One must also bear in mind that this arcus senilis originated through deposits of fatty substances from the capillaries of the limbus similar to the cases of experimental arcus senilis recorded in the publications of Rohr-

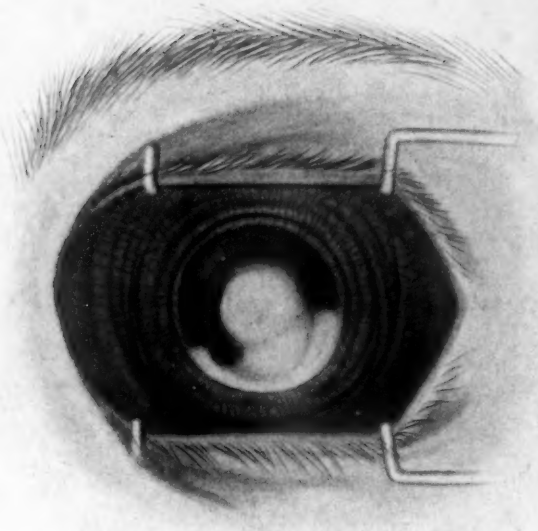

Fig. 15 (case 4).—On the day of admission: The leathery bulbar conjunctiva with its numerous concentric folds shows the typical dark-red color. (The Bitot's spots are purposely omitted.) The cornea shows the superficial ulcer, below the posterior abscess of the cornea and below the semifluid hypopyon in the anterior chamber.

schneider[40] after overfeeding rabbits with a mixture of fats and cholesterin. De Gouvêa, as a matter of fact, mentioned the appearance of arcus senilis in some of his cases, saying nothing, however, about the fate of this formation; according to his description, these were broad, clouded belts in the corneal periphery without clear intervals, which might have been the same white coloration of the limbus that I observed in cases in which the xerosis covered the corneal margin. This may also have been the condition described in one of the first reports on keratomalacia in children, in 1836, by Rau, as quoted by de Gouvêa: "The corneal periphery shows genuine arcus senilis. Elevations formed on this circle, in which no vessels could be distinguished even with a strong magnifying glass." I have no doubt that these "elevations"

Fig. 16 (case 5).—On the day of admission: An incised skin abscess and a genuine abscess of the region of the right nipple are present. Notice the scaly texture of the skin.

which Rau had in mind were xerotic deposits in the cornea, of the kind mentioned in cases 2 and 3, and that this was a genuine xerosis of the limbus and not a gerontoxon formed, as in the present case, after regression of the acute symptoms in the depth of the cornea, and indistinguishable from true gerontoxon.

Case 5.—A coolie, aged 45, was admitted to the hospital on March 31, 1928. He had been ill for three or four weeks, but neither he nor his companion was able to describe the early symptoms. For two weeks he had been blind in both eyes, but there had been no pain. During this period, abscesses appeared in various parts of the body, also without pain. He refused to be admitted to the

40. Rohrschneider: Experimentelle Untersuchungen über die infiltrative. Verfettung der Cornea beim Kaninchen, Graefe's Arch. f. Ophth. **115**:535, 1925.

hospital by the eye department and was referred to the surgical clinic, where several of the abscesses were incised. Four days later, he reappeared and was admitted. According to his statement, the loss of sight was not preceded by any inflammation or secretion worth mentioning.

Physical Examination.—The patient was greatly undernourished. The eyes were deeply sunken. The hair was dry and rough.

The skin was a curious grayish yellow and dry; turgor was absent. Folds formed with the fingers remained a long time. An enormous quantity of wrinkles and many deep folds were on the face. The skin of the entire body was covered with scales, which in certain parts could be removed in large plaques. The skin under the scales was lighter but rather dry. When the hand was rubbed over the body, the scales fell away like bran.

Fig. 17 (case 5).—Three days after admission: Corneal ulcers, abscesses in the skin of the eyelids, and folds and a scaly condition of the skin of the entire face are noted.

Minute exfoliations were present in many places. There were about thirty peculiar abscesses with no signs of inflammation in the vicinity and without pain. A yellowish-gray liquid secretion which generally spread in a downward direction from the mouth of the abscess was found in large pockets. The undermining of the skin often reached a depth of 2 cm. and gave the impression of simple decay. The edges of the opening of the abscesses were curiously rounded and macerated by the oozing discharge, but were not inflamed. Three of these abscesses had been opened in the surgical clinic. The abscesses were found principally on the skin of the temples, on the upper and lower lids and on the chest and abdomen, but there were some also on the skin of the extremities. Figure 16 shows an incised abscess extending medially from the right nipple, and a skin crater, not incised, above the nipple. The scaly texture of the skin is also visible to a certain degree in this picture. In figure 17 are visible the wrinkles of the facial skin, the lighter plaques, which occasionally changed to small exfoliations, deep decay of the skin on the right lower lid, and less marked decay on the center of the left lower lid, left upper lid, and the temporal side, outside of the speculum. The incised

abscesses showed little inclination to heal after the surgical treatment; but after proper nourishment with food rich in vitamin A they disappeared within a week, together with the other abscesses, by the simple process of drying up. The skin pockets then began to adhere and healed in a surprisingly short time.

In the smear from the abscess of the skin, *Streptococcus hemolyticus* was isolated once and *Staphylococcus albus* twice. The blood culture gave negative results.

The condition of finger and toe nails was striking; they were dry, dull, superficially peeled and lined with superficial grooves. The chalky whiteness of the nailbed was most conspicuous and extended to the free edge of the nail. All these symptoms of the skin and nails disappeared entirely after a fortnight's stay in the hospital.

The temperature remained above 38 C. (100.2 F.) during almost the entire period (fig. 18), rose to 39 C. (102.2 F.) and continued at this point, except for slight variations, up to the time of discharge. No cause for this temperature could be found by the department of medicine.

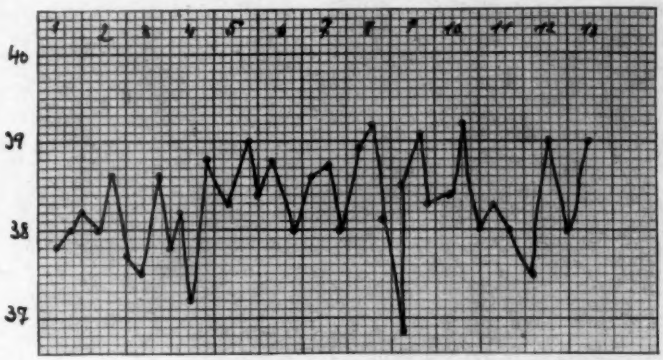

Fig. 18 (case 5).—Temperature curve.

In contrast to the majority of the other patients with keratomalacia, all of whom had diarrhea, this patient suffered from severe constipation, beginning with the third day of his stay in the hospital.

Eye Examination.—On admission to the hospital, the patient showed no photophobia and no lacrimation, in spite of the serious damage to both eyes. The eyes were dry and the discharge of tear fluid was hardly recognizable. There was some dried-up pus and yellow secretion in the angles of the lids. The upper lids were puffy and edematous.

The conjunctivae of the lids were dry, inflamed and uneven, with peculiar irregular folds reaching to the fornix, but with no scars. It was not possible to judge at that time whether or not there was any old trachoma. Both fornices showed dark gray pigment.

The bulbar conjunctiva was reddish purple, slack and dry, very different from an injection of the conjunctiva caused by inflammation. The red hue from fornix to cornea was uniform and it was barely possible to distinguish separate vessels. The entire conjunctiva was thickened, and elevated toward the corneal margin from which it was separated by a step. The bulbar conjunctiva formed rolls round the cornea and several concentric folds, especially near the plica semilunaris, Bitot's spots were not present, although there was general dryness and leathery consistency of the conjunctiva.

With the exception of a marginal zone, of 1.5 mm. in breadth, the right cornea had become one huge ulcer. Its center was whitish yellow, projecting and surrounded at the edges by a slightly raised, dense white infiltration. At this point, the crater-shaped ulcer remained distinct from that part of the cornea which was not yet destroyed (fig. 19). There was no xerosis of the cornea, and no vessels were visible; the whole condition looked like an ulcus serpens.

The left cornea had completely melted and was transformed into a yellowish-white necrotic stopper. Even the limbus was destroyed at five different points (fig. 19), and in certain places was completely covered by the circular fold of the swollen conjunctiva of the eyeball. At 4 and 8 o'clock a dark color was seen that was caused by the already prolapsed iris. Neither the anterior chamber nor the iris was visible in either eye. The tension was —1 in both eyes. Scraping from the cornea and from the conjunctiva gave large quantities of xerosis bacilli and pneumococci that were sprouted in great numbers on the dead epithelium and mucous masses. Some staphlyococci also were present.

Atropine was used for treatment. The conjunctival sac was cleansed by rinsing with a normal physiologic solution of sodium chloride. After two days' treatment, a diet was prescribed that was rich in vitamins, including butter, liver, cod liver oil and lemon squash, and no further local treatment was given.

Fig. 19 (case 5).—On the day of admission: The ulcer of the right cornea, which is still separated from the limbus and the ulcer of the left cornea which reaches to five different points of the limbus, are seen.

The Wassermann reaction was negative. The stool was normal except for *B. coli* and *Ascaris*. Culture of the abscess of the skin yielded *Streptococcus hemolyticus* once and *Staphylococcus albus* twice. The blood culture gave negative results. The urine was straw-colored and cloudy, and contained enormous quantities of phosphates. There was a sediment of epithelial cells from the bladder. The temperature is shown in figure 18. The surgical examination revealed multiple abscesses of the skin. The dermatologic examination suggested erysipelas on account of the decay of the skin. The medical examination gave negative results, and no cause for fever could be found. Examination of the blood showed: 3,800,000 red blood cells, 25,000 white blood cells, polymorphonuclears 90 per cent, lymphocytes 10 per cent, and hemoglobin 65 per cent (anemia).

Course of Disease.—April 4, 1928: After two days of a diet with plenty of vitamins, secretion of the eyes began; the ulcers became shiny, and the scabs of the bulbar conjunctiva and of the left cornea fell off, so that perforation was plainly visible. The conjunctiva of the lid and that of the eyeball were a dull bluish red, though somewhat less dry than before. The corneal ulcers still projected but did not advance any further toward the limbus.

April 7: The bulbar conjunctiva and conjunctiva of the lid of the right eye were shiny, and the injection was a brighter red. The edges of the right corneal

ulcer were also shiny and more sharply defined; the ulcer cleared up quickly, so that perforation seemed unavoidable.

The upper lid of the left eye was swollen. The patient began to be in pain. The iris prolapsed into the ulcer in the form of a dark ring. The conjunctiva surrounding the cornea was swollen, coarse and chemotic. Movements of the eye were painful and limited; a beginning panophthalmitis. The patient refused the suggested evisceration of the eyeball.

April 9: The periphery of the cornea of the right eye was strikingly clearer, and the ulcer, sharply defined and perforated in the lower half, appeared somewhat smaller. The iris was lying partly in the ulcer.

Much pus had been discharged from the interior of the left eye, the chemosis had decreased, the pain had almost disappeared, and the corneal ulcer was sunken.

April 10: During an attempt to give the patient a closer examination in the clinic, he collapsed and became unconscious.

April 12: The patient demanded that he be allowed to return home as he felt better. His general condition had visibly improved; he appeared brighter, his skin was partly shiny and smoother, and the abscesses had healed; the nails, however, were still grooved, although the chalky whiteness had disappeared. His temperature was still 39 C. (102.2 F.). Examination of the right eye showed a central regressive corneal ulcer with minor paracentral prolapse of the iris in the process of healing. No vessels were visible. The lid and conjunctivae of the eyeball were much injected but shiny. There were no signs of xerosis. In the left eye, there was a beginning phthisis bulbi. Vision in the right eye was reduced to uncertain projection and light perception at a distance of 5 meters.

Comment.—This was by far the worst case of the six. In view of the still more severe nature of the general symptoms, the disease of the eye, in itself extremely severe, retired somewhat into the background. The disease represented that type of keratomalacia or rather that stage of keratomalacia in which it would be impossible to make a diagnosis without knowledge of the foregoing cases; which would either remain forever undecided or in which the diagnosis keratomalacia, made without comprehension and consideration of the entire picture, could never be more than a conjecture.

At the beginning, the eyes were so covered with yellow, dry crusts that nothing was to be seen either of a xerosis of the conjunctiva or of the cornea. Even after removal of the crusts of the bulbar conjunctiva nothing was visible but an irregular purplish gray conjunctiva of the fornix and of the eyeball, which projected from the palpebral fissure in leathery folds, in sharp contrast to the conjunctiva of the lid which had, relatively speaking, changed very little. After the yellow crusts, which also adhered firmly to the cornea, had been removed, there was again in the right eye a picture like a severe ulcus serpens, or a perforated gonorrheal ulcer, similar to the use in case 4. In the left eye the destruction extended into the limbus and at one point even into the sclera, a condition rarely seen in ulcus serpens which is generally arrested at some distance from the limbus. The boundary between the ulcus and the remaining cornea in the right eye was sharply defined, the center of the ulcus projecting; the ulcers were so perforated in the left

eye, that the iris, dark and covered with pus, became visible. Frequent irrigations with normal physiologic solution of sodium chloride, the use of atropine and general nourishment arrested the ulcer of the right eye after a small perforation at one point on the eighth day; an infection penetrated the left eye, forming an abscess of the interior of the eye, which fortunately, as the patient refused evisceration, discharged of its own accord. Shrinking of the eyeball followed.

The general symptoms were, however, of great interest and still more important than the local symptoms. The patient, though only 45 years of age, looked like a man of 60 and gave the impression of mental confusion. The face and body were a curious dark grayish brown. The skin lay in small wrinkles and deep folds, especially on the face; it was very dry and earthy, and the patient's bed was always covered with scales. The hair also was dry, faded and rough, and was falling out. The skin of the trunk as well as of the extremities fell apart like tinder and developed a secondary infection which was the cause of the numerous abscesses, containing different germs. In spite of this there was not much sign of inflammation anywhere on the skin and no swelling round the abscesses. In fact, the whole organism was in such a state of intense exhaustion that it was incapable of self-defense by means of inflammation. That the abscesses also were caused by the main disease was proved by the successful results of the vitamin nourishment which cleared up the abscesses immediately without any surgical treatment, although skin pockets, 2 cm. in depth, had formed. Those abscesses which had been opened by surgical methods showed little inclination to heal until after the treatment by means of nourishment had been introduced.

The changes in finger and toe nails should also be mentioned. The nails were grooved from the base of the nail to the outer edge, which had started to crack, and were a chalky white from the bed of the nail to the edges. All these changes had completely disappeared at the time of the premature discharge of the patient.

In contrast to the other patients, this man suffered from severe constipation. Bacteriologic examination of the stool showed only *Ascaris* and *Bacillus coli*. The urine was curiously light and cloudy and had masses of exfoliated epithelium in the sediment.

The blood showed a great decrease in hemoglobin and a moderate decrease in red blood cells and leukocytosis of 25,000. The polymorphonuclear content was 90 per cent, and the lymphocyte 10 per cent. The temperature remained between 38 and 39 C. (100.4 and 102.2 F.), and was still at this height at the time of discharge. No cause could be found for this rise by medical examination.

After thirteen days in the hospital, the patient was able to distinguish a ray of light in front of the right eye, and felt subjectively so much better that he demanded to be allowed to go home. Since then he has

not reappeared. The degree of the general disturbances caused by the disease was proved not only by the mental confusion noticed in the beginning, but also by the collapse, two days before discharge, when the patient was trying to stand up for the purpose of further examination.

CASE 6.—A silversmith, aged 25, was admitted to the hospital on May 22, 1928. The disease had started in both eyes simultaneously twenty days before with burning of the eyes but without redness. After a few days, the decrease in vision was rapid. For seven days preceding admission the patient had been blind. There was a little secretion and a dry feeling in both eyes with occasional mild pain, but there were no other subjective symptoms. For seven days he had been taking a Chinese medicine.

Examination.—On admission, May 22, 1928, the patient showed a painless fluctuating abscess the size of a hazelnut in the region of the right eyebrow. There were also abscesses between the thumb and first finger of the right hand, on the chest and back, the left thigh and the right buttock. The skin was dry, the face bloated and the hair lifeless.

There were dry crusts on the margins of the lids, which were, however, not inflamed. Only a slight secretion was present in the inner and outer angles of

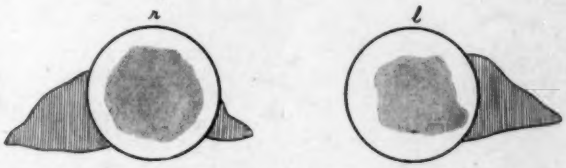

Fig. 20 (case 6).—On day of admission: Large Bitot's spots of the temporal sides of the conjunctivae of both eyeballs are present. Both corneas show the size of a serpent ulcer-shaped central ulcer which soon perforated.

the lids. The tear ducts were normal. The conjunctiva of the lids of both eyes was slightly uneven and reddened. The lower fornix had stiff, dark gray folds which were mistaken for argyrosis, in the beginning, on account of the patient's occupation of silversmith, but which was the typical discoloration of the conjunctiva in keratomalacia seen in cases 1, 2, 4 and 5. There was no papillary hypertrophy. The conjunctiva of the eyeball was grayish red and dry, with numerous dry folds directed concentrically to the limbus. In the region of the palpebral fissure were two large triangular Bitot's spots with wrinkled conjunctiva. A red belt of dried conjunctiva surrounded the cornea.

The cornea of each eye had completely lost its luster right up to the limbus, and was dry. There were no signs of xerotic whiteness, merely an opaqueness. Both corneae were occupied by circular, central ulcers, which as first sight appeared to be ulcus serpens (fig. 20), but no hypopyon was present. The ulcus was gray in the center, and the margin was surrounded by a white double ring, in sharp contrast to and sharply separated from the gray marginal zone of the cornea. No blood vessels were visible. The center of the ulcer was slightly projected, but as yet there was no sign of perforation. The diameter of the ulcers of both eyes was about 7 mm. The anterior chamber, iris and lens were not visible.

Surgical examination showed abscesses in various parts of the body which were considered syphilitic. Dermatologic examination revealed a nongonorrheal urethritis of indefinite origin. The Wassermann reaction was negative. The urine was

clear, alkaline and straw-colored, with a specific gravity of 1.020. Examination of the blood showed: 4,800,000 red blood cells, 18,000 white blood cells, polymorphonuclears 90 per cent, lymphocytes 10 per cent and hemoglobin 65 per cent. On two examinations the stool was normal. Bacteriologic examination of scrapings from the cornea gave *Streptococcus hemolyticus, Pneumococcus* type IV and *Staphylococcus aureus.* The temperature was about 38 C. (100.2 F.) during the first six days after admission, after which it was about 37 (98.6 F.) (fig. 21.). The basal metabolism showed: basal metabolic rate + 15.6 per cent; calories per day 1,579, and respiratory quotient 0.727.

Course of Disease.—May 29, 1928: Perforation of the center of both ulcers, which had not continued to advance toward the margin, had occurred. The iris, seen as an indistinct dark mass in the center of the cornea, was still covered with fibrin. The margin of the ulcers had been reached by a few deep vessels from different sides, but not in any large number. The marginal parts of the cornea had cleared up, and small spherical globes which were taken to be edematous vesicles could be seen by means of the corneal miscroscope at the edge of the ulcer, an indication that the tissue had regained some of its dampness. The treatment

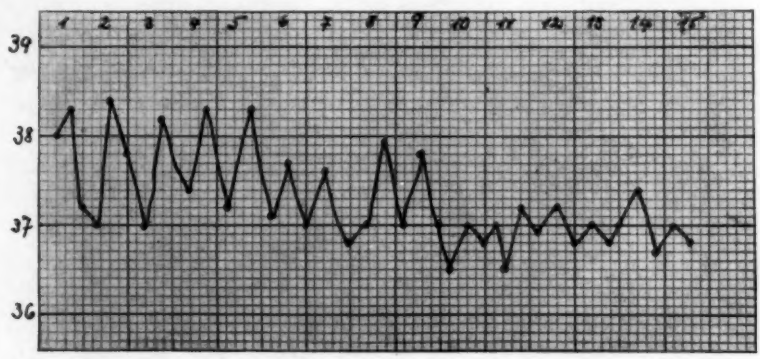

Fig. 21 (case 6).—Temperature curve.

prescribed was nourishment rich in vitamin A and cod liver oil. Except for the use of atropine, there was no local treatment.

June 25: Both ulcers had healed since the examination of May 29. The condition now was leukoma adhaerens. Optical iridectomy was attempted in the right eye at 12 o'clock, the only point at which any anterior chamber remained. A week later, a small iris staphyloma was leveled in the left eye by means of thermocautery. This also healed perfectly. Vision in both eyes was reduced to good light perception and projection. The entire condition of the patient had changed. The texture of the skin was now normal, the hair had its normal luster and the abscesses had completely healed.

Comment.—This man, aged 25, was admitted to the hospital at a stage when both corneas had been destroyed in the course of twenty days by an ulcer similar to ulcus serpens. This is the late stage of keratomalacia seen most often in the clinics in China. The marginal parts of the cornea were opaque and dry, but there were no white xerotic deposits on the cornea as seen in cases 1 and 2, except the Bitot's spots on the conjunctiva of the eyeball. Perforation of the ulcers took place soon

after admission, but it was possible to execute an optical iridectomy on the right eye which showed a leukoma adhaerens. In the case of the left eye, it was necessary to cauterize a small pinhead prolapse. The diagnosis keratomalacia was only possible in view of the extensive xerosis (Bitot's spots) and the wrinkling of the eyeball conjunctiva; the discoloration of the conjunctiva of the fornix, with its elevated little folds, was so dark a brown that argyrosis of the conjunctiva was at first considered, especially on account of the patient's occupation of silversmith. Professional argyrosis, however, could not be proved, and there was no argyrosis of the cornea. This discoloration was connected with keratomalacia or rather with the general disease underlying keratomalacia, especially as it disappeared under our observation after proper nourishment. The yellow discoloration of the conjunctiva of the eyeball, which might easily have been mistaken for imbibition with blood, also faded away completely.

Among general symptoms, the curious dryness and discoloration of the skin and hair and the multiple abscesses without inflammatory symptoms should be mentioned. In ignorance of the disease of the eye, these were diagnosed as syphilitic abscesses by another department, because of the unusually livid discoloration and the presence of similarly discolored crusts. The Wassermann reaction of the blood and cerebrospinal fluid, however, was negative, and all other symptoms of syphilis were absent. All of the abscesses healed as soon as proper nourishment and cod liver oil were given, as did also the process in the eye. It should be especially emphasized that the corneal ulcers showed a marked inclination to diminish in size; the final scar, therefore, proved far smaller than had been originally expected. The edges of the scar which were also dense in the beginning, cleared up to a most unusual extent in a short time. The same extensive disappearance of the opacity was observed in case 4.

The red blood cells were normal, but there was a leukocytosis of 18,000 with 90 per cent leukocytes and 10 per cent lymphocytes. The hemoglobin content was distinctly lower, 65 per cent.

Mention should also be made of a curious discharge from the urethra which this patient showed. No gonococci were found in spite of prostatic massage, but the smear showed numerous epithelial cells, which seemed superabundantly exfoliated, resembling the increased peeling of the outer skin and of the xerotic process of the conjunctiva. Further research in this direction will show whether the epithelial apparatus of these regions is influenced by the main disease and whether it experiences the same changes as the conjunctiva of the eye.

This patient remained in the hospital for a period sufficiently long to observe the disappearance of all the general symptoms and the healing of the keratomalacia without any attempt at local treatment.

(To be continued)

THE OCULAR PATHOLOGY OF PERIARTERITIS NODOSA *

ISADORE GOLDSTEIN, M.D.
AND
DAVID WEXLER, M.D.
NEW YORK

The term periarteritis nodosa was given by Kussmaul and Maier,[1] in 1866, to a distinct pathologic entity characterized by the presence of nodular thickenings of various sizes in the walls of small and medium size blood vessels and caused by inflammatory disease. The disease is not rare. About 120 cases have been reported and the various aspects of the condition fully elucidated in this country by Longcope,[2] Klotz [3] and Ophüls;[4] and abroad, rather voluminously, by Gruber.[5]

While the lesion which Kussmaul and Maier described has remained established as a separate entity, it has become apparent through study of successive cases that it is in reality an arteritis or panarteritis involving, as it usually does, all the layers of the wall of the artery. Further, it is clear that macroscopic nodes are entirely absent in some cases. The lesion is then found to affect only the small arteries, which nevertheless show microscopic periarterial nodes of inflammatory reaction.

The evidence of competent observers indicates the lesion to be of a distinctly inflammatory character, and its histologic characteristics to be somewhat as follows:[6] One of the characteristic features in the early cases is a tremendous infiltration of the adventitia with neutrophilic and eosinophilic leukocytes, with a smaller or larger proportion of small lymphocytes and plasma cells. There may be early severe degeneration of the media leading to destruction of the muscle, so that the arteries may rupture and hematomas or false aneurysms develop. Usually the changes in the media are moderate and slower to develop. Leukocytes soon invade the media, and in turn the intima. In some

* Submitted for publication, May 25, 1929.
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* From the Laboratories and Ophthalmological Division of the Mount Sinai Hospital.

1. Kussmaul and Maier: Deutsches Arch. f. klin. Med. **1**:484, 1866.
2. Longcope: Bull. Ayer Clin. Lab. Penn. Hosp. **5**:1, 1908.
3. Klotz: J. M. Research **164**:1, 1917.
4. Ophüls: Periarteritis acuta nodosa, Arch. Int. Med. **32**:870 (Dec.) 1923.
5. Gruber: Virchows Arch. f. path Anat. **258**:441, 1925.
6. Ophüls (footnote 4).

cases, the latter remains practically unaltered; in others, it is soon found to contain masses of fibrin and hyaline material. Further changes consist in the invasion of adventitia and diseased media by epithelioid cells, the formation of new capillaries and fibrous tissue, and proliferation of intimal cells. Aneurysmal formation depends on the extent of destruction of the muscle and the resistance of the new fibrous tissue. The prominent nodules are usually aneurysms solidified by the organization of a thrombus in them.

The disease seems to have a predilection for the mesenteric, renal, hepatic, pulmonary and cerebral vessels, as well as vessels in the

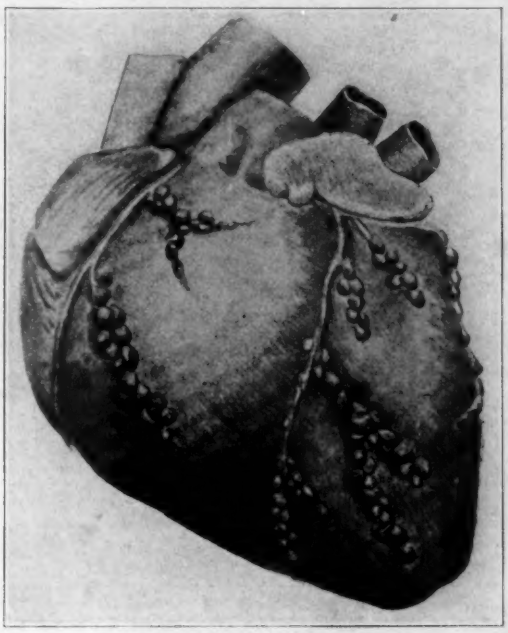

Fig. 1.—Periarteritis nodosa of the epicardium (from Guldner: Virchows Arch. f. path., Anat., 1914, vol. 219).

skin and muscles of the extremities. The symptoms correspond to the effects of the development of thrombosis, aneurysmal dilatation and rupture of arteries on the respective organs. Myocardial fibrosis from disease of the coronary arteries, multiple hemorrhagic renal infarcts with occasional acute or subacute hemorrhagic nephritis, multiple small aneurysms and ruptures of cerebral arteries leading to areas of softening, and peritonitis from disease of the mesenteric artery or its visceral branches—these are the more frequent manifestations of the disease.

Clinically, the disease is found at almost any age, although young persons are usually selected. The usual course is a chronic sepsis, with great emaciation, anemia and an irregular temperature curve. Definite periods of remission and exacerbation have been observed, indicating a tendency to healing. Other symptoms depend on the organs affected; when these are multiple, as is usual, the symptoms are so numerous and the clinical picture so bizarre as to render correct diagnosis during life difficult, and indeed infrequent. A biopsy of a local nodule may help. Baehr and Manges [7] reported a diagnosis

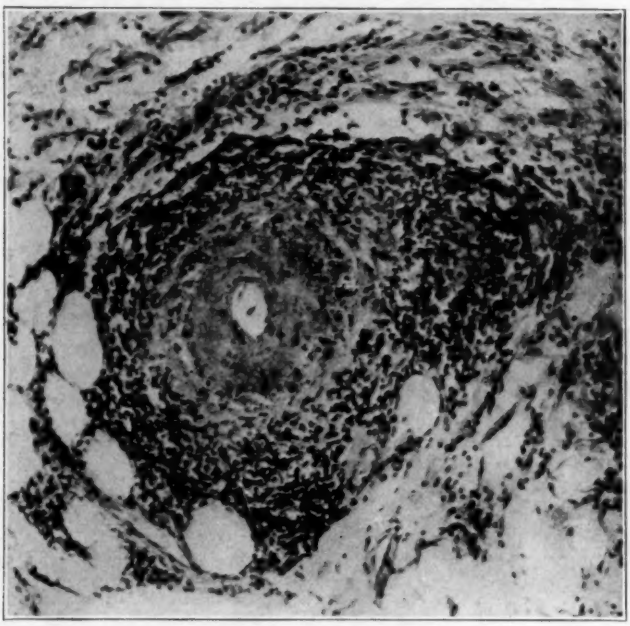

Fig. 2.—Small artery of the epicardium showing necrosis of the media and dense adventitial infiltration with polymorphonuclears and round cells.

made from a mesenteric nodule excised during a laparotomy for symptoms of acute appendicitis. Gruber found a nodule in the cystic duct in a patient operated on for acute cholecystitis.

In the etiology, syphilis, long suspected as being a factor, has been virtually excluded. Rather than being due to a specific agent, the most widely accepted view is that it is merely a symptom of a generalized infectious or toxic process for which no single organism can be held responsible.

In spite of repeated observations of the fundus in proved cases of periarteritis, the eye remains the sole vascular organ in the body in

7. Baehr and Manges: Am. J. M. Sc. **162**:162, 1921.

which the lesion and its effects have not been fully described. The sole reference in the literature is that of P. Müller [8] in 1899, whose brief description follows:

The same changes were found in the retinal arteries as in the small vessels of the brain. All degrees of blood vessel disease could be made out. Arteries with mild inflammatory change in the adventitia were close to others in which the whole arterial wall was infiltrated with leukocytes and fibroblasts. In addition, fibrinous exudate in the media with necrosis of the latter were seen. Also, proliferation of the endothelium of the intima was present, but not to a marked degree. In the vicinity of the diseased vessels was a variable amount of edema.

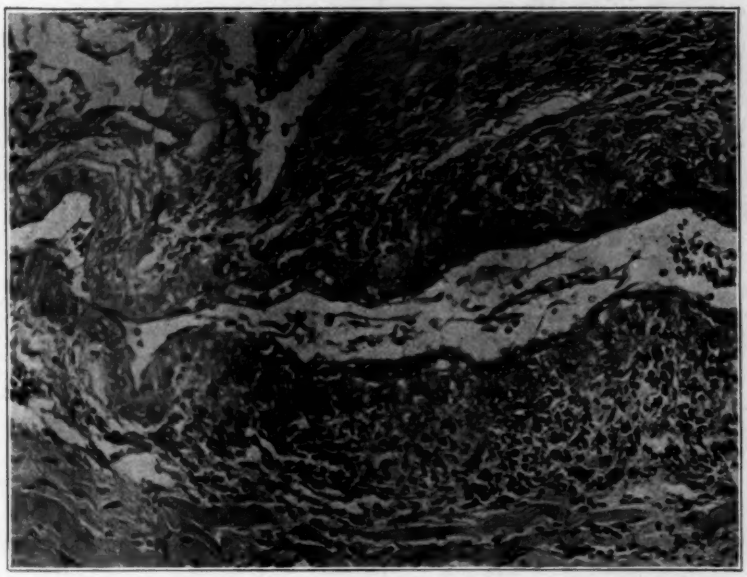

Fig. 3.—Medium sized artery of the diaphragm; the portion to the left is normal; that to the right shows necrosis of the media, and infiltration of the adventitia with polymorphonuclears and round cells.

The case to be reported is of interest not only because it offers the first complete study of the eye changes in this disease, but also because it illustrates the type of case with only microscopic nodes, as well as the close association of the disease with a generalized infective process.

REPORT OF CASE

History.—M. K., an American girl, aged 19, was admitted to the hospital with multiple red, painful and swollen joints, dyspnea and marked weakness. She had been operated on for appendicitis six months before, and had been treated

8. Müller, P.: Festschr. d. Stadt. Krankenh. Dresden-Friedrichstadt, Dresden, 1899, p. 458.

since for symptoms of renal disease and pleurisy. She was acutely ill, pale and dyspneic, with a temperature of 104 F. In addition to signs of mitral disease and pleural effusion, there was evidence of subacute glomerular nephritis. The blood count showed: hemoglobin, 52 per cent; red blood cells, 2,280,000; white blood cells, 10,200; polymorphonuclears, 87 per cent; lymphocytes, 4 per cent; eosinophils, 5 per cent, and mononuclears, 4 per cent. Analysis of the urine showed a specific gravity 1.036 (fixed at from 1.010 to 1.015), and an excessive amount of albumin and casts. The blood urea which was 41 mg. per hundred cubic centimeters of blood, rose to 71 mg. in the next few weeks. Signs of pericarditis developed. The hemoglobin dropped to 39 per cent. The Wassermann test and repeated blood cultures yielded negative results.

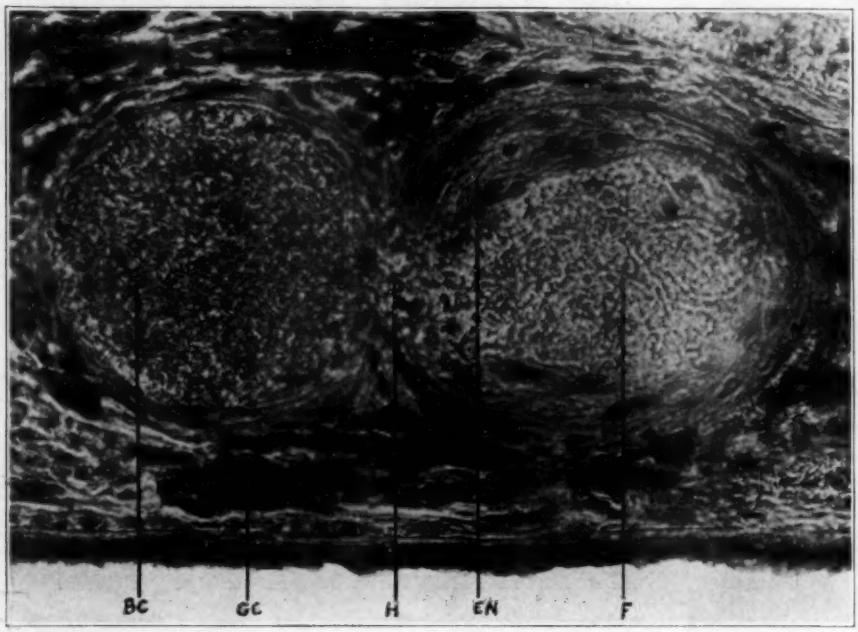

Fig. 4.—Two choroidal arteries, showing early necrosis, *EN*, and fibrinoid changes, with swollen endothelial and medial cells; *H*, hemorrhage in wall of artery; *GC*, giant chromatophore; *BC*, blood clot, and *F*, fibrin clot.

On the basis of the pain in the joints and the cardiac and renal disease with anemia, a general infection caused by a rheumatic virus was considered. The course, except for a slight remission, was steadily downward, with irregular temperature, vomiting of blood, exacerbation of pain in the joints and the onset of abdominal pain. Death due to cardiac collapse following bronchopneumonia occurred four months after admission.

The fundi, examined on only one occasion, were free from abnormality.

Autopsy.—There was considerable edema of the legs and feet. Throughout the skin were many fine discrete hemorrhages the size of a pinpoint, and a few purpuric spots.

The cortex of the brain was covered with a gelatinous, edematous exudate under the pia-arachnoid.

Both pleural cavities contained considerable straw-colored fluid. The lungs had many fine adhesions to the wall of the chest. There were a number of circumscribed hemorrhages on the pleural surface of the lungs. Section showed numerous dark areas of hemorrhage. The bronchi were normal, the pulmonary vessels dilated.

The pericardium was thick and adherent, its surface studded with minute hemorrhages. There was a soft, adherent vegetation with a greenish base on the mitral valve, also a number of verrucous lesions along the valves. The aorta and coronary arteries showed no changes. The myocardium showed a few flecks the size of a pinpoint.

The viscera of the abdomen were pale; there was about 1 liter of clear, straw-colored fluid.

The liver was extremely pale and showed considerable fatty degeneration of the periphery of the lobules on section.

Both capsules of the kidneys were thick and edematous. Their surface was covered with numerous hemorrhages causing a marbled mottling. There were a

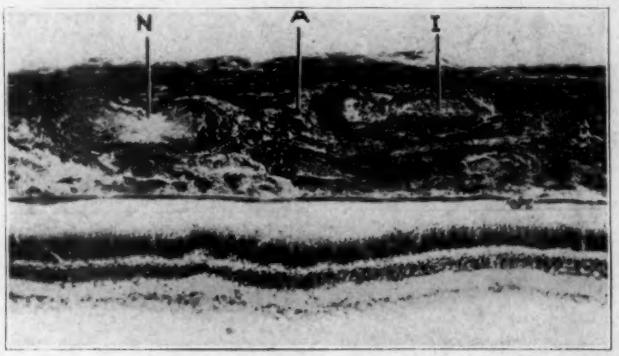

Fig. 5.—Section of the choroid and retina; two choroidal arteries; *N*, necrosis of media; *A*, adventitial and periadventitial infiltration, and *I*, intimal proliferation.

number of old and fresh fibrotic scars. Section revealed extensive hemorrhages throughout the cortex and the medulla. The latter were distorted in their relations. In the cortex were deep yellow infarcts, while in the pylovenous angle of one of the calices a thrombosed vein was observed.

The stomach was dilated, covered with mucinous exudate and showed many flecks of hemorrhage the size of a pinhead, which on close view were seen to cover superficial ulcers of the mucosa.

The uterus was normal.

The microscopic study revealed the following conditions:

The alveoli of the lungs were filled with exudate, their walls congested and infiltrated with polymorphonuclears. The bronchi contained pus.

The muscle fibers of the heart showed parenchymatous degeneration; the small arteries showed arteritis.

Most of the glomeruli of the kidney were normal. Some showed more or less advanced stages of fibrosis. A few showed endothelial proliferation, with collapse and fusion of the loops. Several interlobular arteries had canalized thrombi in their lumen. There was beginning organization of a thrombus in a large arcuate artery, with adventitial infiltration and a wide zone of peri-

adventitial fibrosis. In the medulla were areas of hemorrhage and necrosis. Acute arteritic changes were absent.

Careful examination of the brain failed to reveal change in the tissues or arteries.

The diagnosis was: periarteritis nodosa; subacute verrucous endocarditis (mitral and aortic); subacute glomerulonephritis; multiple infarcts of the spleen and kidneys; fatty degeneration of the liver; chronic fibrous pericarditis and

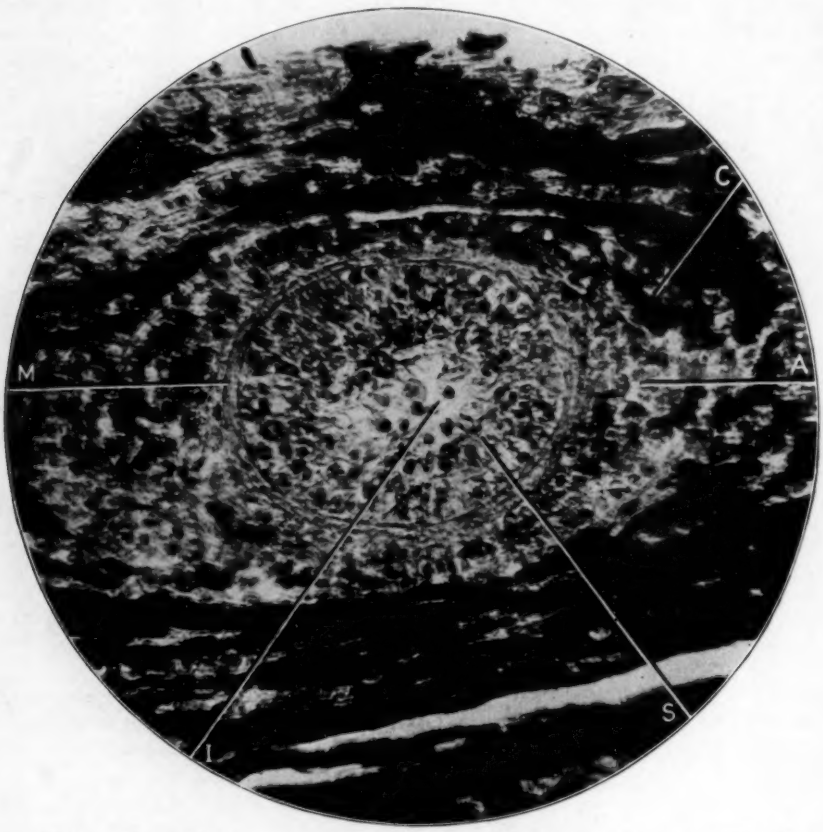

Fig. 6.—Choroidal artery showing uniform dilatation; *I*, intimal proliferation; *S*, increase of subintimal tissue with infiltration of the inner portion of the media; *M*, piston-ring shaped remains of media; *A*, adventitial and periadventitial infiltration, and *C*, giant chromatophore.

pleuritis; anasarca; focal myocarditis; petechial hemorrhages in the skin; pulmonary edema and diffuse hemorrhage.

Microscopic Examination.—Microscopic study of the ocular structure showed the following facts: The vessels of the iris and ciliary body were normal; no change was seen in the tissues. The central retinal artery and the retinal arterioles, as far as they could be traced, showed no change. The ciliary nerves were normal as was the nerve head.

The changes in the structure and the vessels appeared to be confined strictly to the choroid. The capillary layer and the veins were of the usual size, but the great majority of the arteries were enlarged circularly or longitudinally and showed inflammatory changes in varying stages and degrees.

Some exhibited early changes in the form of a fibrin clot with a few degenerated blood cells, early necrosis of the inner media and swelling of the endothelial and medial cells (fig. 4). Others, more diseased, showed rich adventitial

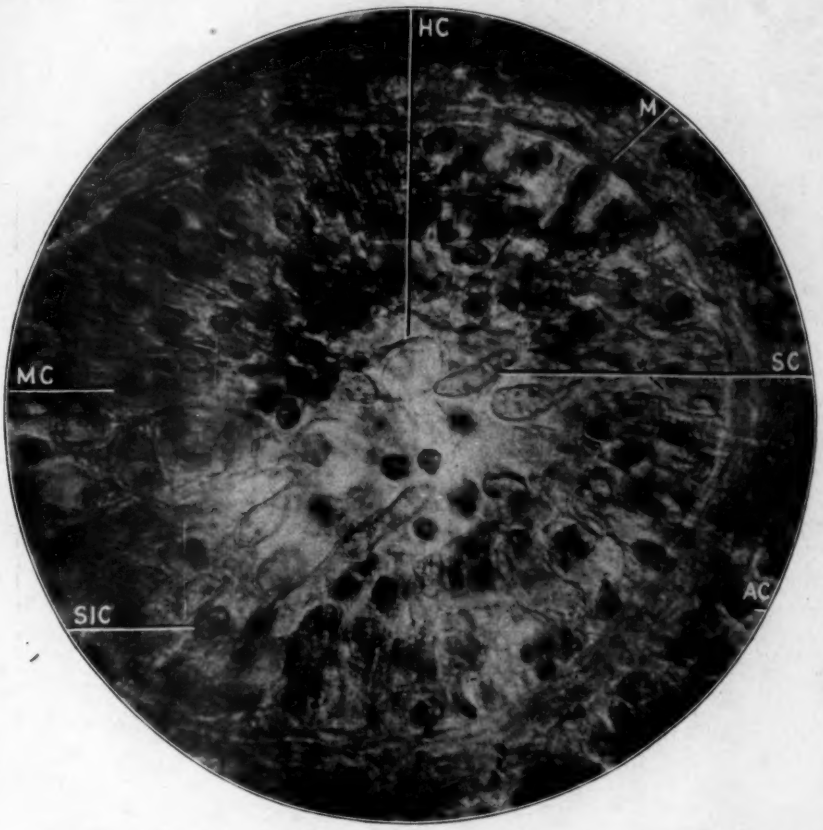

Fig. 7.—Oil immersion study of the choroidal artery of figure 8; *HC*, intimal cell undergoing hyaline degeneration; *SC*, hypertrophied spindle cell of intima; *SIC*, cell of proliferated subintimal tissue; *MC*, infiltrating cell of media; *M*, piston-ring media, and *AC*, cell of infiltrated adventitia.

and medial infiltration, varying degrees of intimal proliferation without or with thrombosis (figs. 6 and 11, respectively); and in some cases necrosis and fragmentation of the medial wall (figs. 5 and 10). Some exhibited a media which, though markedly infiltrated, was nevertheless intact (fig. 5, right). In others, the media was largely replaced by infiltrated cells or by proliferated subendothelial tissue (fig. 6). Figure 5 illustrates a portion of the choroid containing two horizontally cut arteries, with medial and adventitial infiltration. Both media were

about equally infiltrated, but that of the vessel on the left was fragmented and severely necrotic.

Figure 6 represents an artery showing typically severe inflammatory change. The vessel was enlarged, occupied almost two-thirds the width of the choroid, and together with the surrounding infiltration displaced the choroidal pigment. The lumen was almost obliterated through proliferation of endothelial cells. The limits of the latter could not be defined, but the subendothelial tissue was hypertrophied to three or four times its normal width. The cells nearer the center were spindle-shaped, had a faintly staining cytoplasm, and were disposed radially (fig. 7); others were vacuolated (cystic degeneration) and larger. Those nearer the muscle coat were polyhedral, with a round, dark-staining nucleus. Some of these were also cystic. Here and there through this layer were fine fibrils. Occasional clumps of pink-staining, coagulated plasma were seen between the subendothelial tissue and the media. The media was represented by a narrow

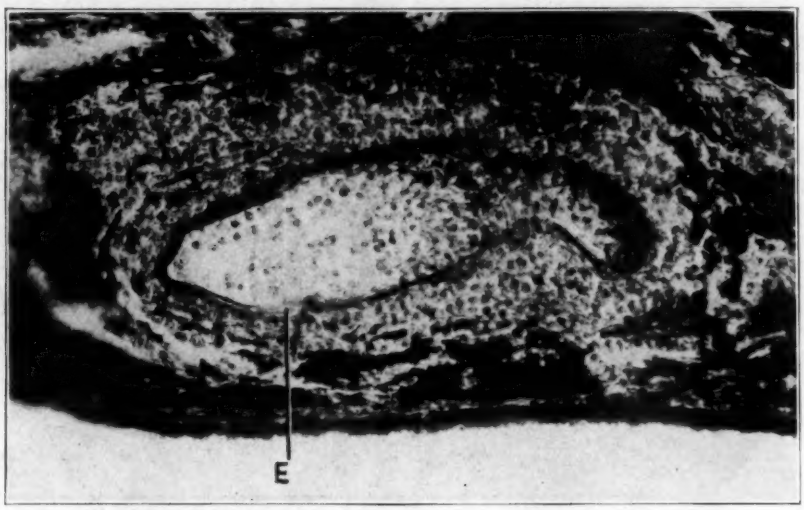

Fig. 8.—*E*, interruption and fragmentation of the elastic lamina of the choroidal artery; Wiegart elastica-van Gieson stain.

circular band, its inner portion having been largely replaced by proliferated subendothelial and inflammatory round cells. Its inner wall was sharply defined, while its outer wall fused with the adventitia. This striking "piston-ring" appearance was found in none of the other organs in this case. The adventitia contained many lymphocytes, a few mononuclears and plasma cells and an occasional polymorphonuclear. The periadventitia was similarly infiltrated.

The adventitial lesion occupied for the most part the sides of the vessel corresponding to the long axis of the choroid, those next the retina and sclera being but little infiltrated. In addition to round cells there were endothelial leukocytes and spindle cells. Of the latter, some were finely granular (partly depigmented) chromatophores; others were nonpigmented fixed connective tissue cells. Free pigment was dispersed through the tissue spaces; some had been taken up by a giant type of cell. The transformation of spindle-shaped chromatophores into giant cells is again shown in figures 4 and 6.

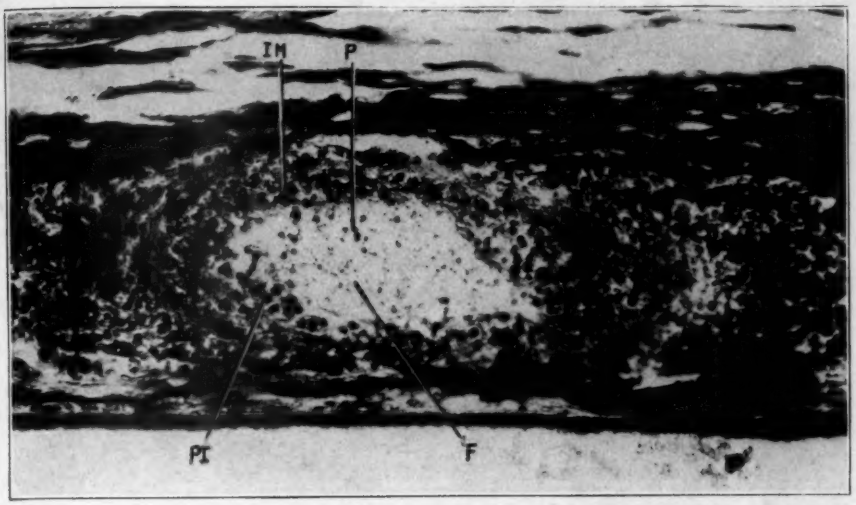

Fig. 9.—Choroidal artery with fibrin clot, *F*, containing degenerating polymorphonuclears, *P*; *PI*, proliferated intima, and *IM*, infiltrated media.

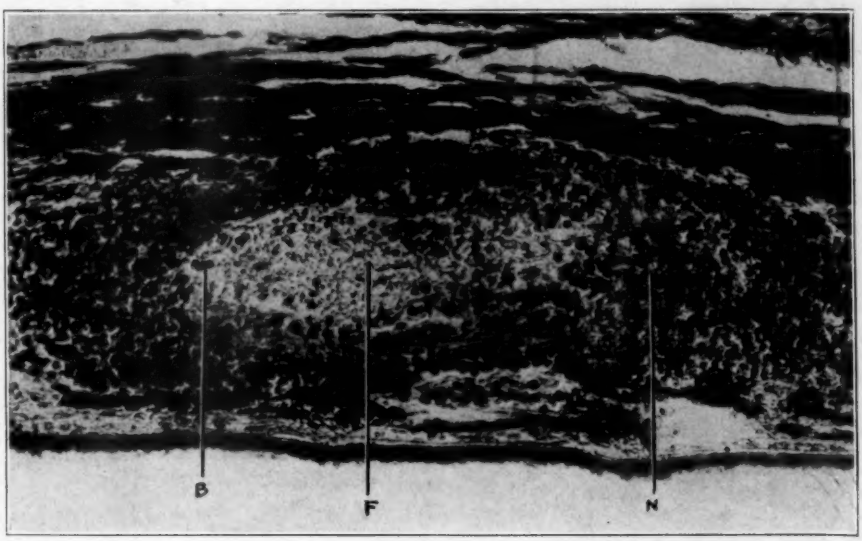

Fig. 10.—Choroidal artery with fibrin clot, *F*, showing early organization with fibroblasts, *B*; severe necrosis of media, *N*, with lymphocytes, endothelial phagocytes, plasma cells and a few polymorphonuclears.

The elastic lamina in some vessels was fragmented or interrupted in its continuity (fig. 8). In others, in the presence of mild changes, it was intact.

The state of the lumen seems to be independent of the degree of intimal change. Figure 4 shows a fibrin clot with early intimal change. Figure 9 shows a similar clot with moderate intimal change; while figure 10 shows a fibrin clot undergoing organization, with marked intimal and medial change.

Figure 11 illustrates complete thrombosis through marked intimal proliferation.

COMMENT

From the presence of fibrin clot in some vessels and marked intimal proliferation in others, the necrosis of the media in some and its preservation in others and the varying state of the elastic lamina, together with the varying degrees of medial and adventitial inflammation, it is reasonable to assume different stages and degrees of inflammatory reaction. However, neither the tendency toward thrombus formation nor the height of intimal proliferation can be related to the degree of arterial or periarterial inflammation.

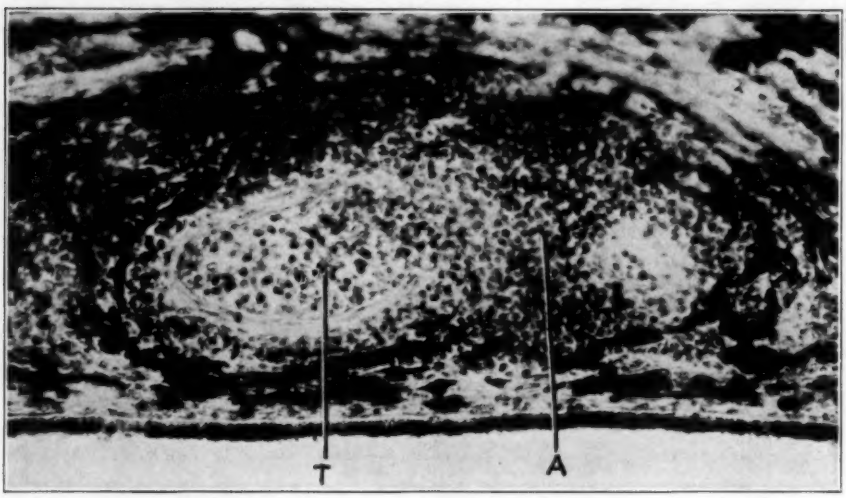

Fig. 11.—*T*, complete occlusion (thrombosis) of the artery by proliferated intimal cells; *A*, adventitial and periadventitial infiltration with round cells (small and large mononuclears, lymphocytes and plasma cells) and a few polymorphonuclears.

The predominating inflammatory cells in the choroidal lesion are of the monocytic variety (lymphocytes, large mononuclears and plasma cells); the predominating type in the arteritis elsewhere in the body is the lymphocyte (figs. 2 and 3). In these organs, however, the arteritis is more acute, the media is swollen, infiltrated and necrotic (fibrinoid degeneration), and the intima is also necrotic but not proliferated. Also, necrosis of the media is the most prominent feature.

These differences point to a less severe and perhaps chronic form of infection of the choroid.

The ophthalmoscopic appearances resulting from multiple choroidal periarteritic nodules in the presence of an apparently normal retina are perhaps not difficult to conjecture. It is possible that they might appear as scattered whitish foci, and might even suggest tubercle formation. As far as our case is concerned, we regret that conclusive studies of the fundus are not available. Of the fourteen cases that Gruber [9] collected in which reports on the fundus were available, practically all showed some degree of renal disease as a complication. Among these, the patients who had developed uremia and hypertension showed albuminuric retinitis. The others exhibited relatively normal fundi. Gruber searched his own case carefully before the onset of renal disease, but he mentioned specifically the absence of choroidal foci. However, that this type of lesion could give rise to or even resemble the picture of albuminuric retinitis is extremely unlikely.

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9. Gruber: Virchows Arch. f. path. Anat. **258**:470, 1925.

OCULAR LESIONS IN TULAREMIA

REPORT OF TWO CASES *

J. HEWITT JUDD, M.D.
BEATRICE, NEB.

In 1911, McCoy and Chapin,[1] while investigating a fatal epidemic of what was supposed to be bubonic plague in the California ground squirrels, isolated a small, gram-negative, nonmotile and nonspore-bearing pleomorphic organism, which they named *Bacterium tularense,* since it was found in Tulare County, California.

During their investigations two of the laboratory workers contracted what was later found to be the human form of this infectious disease. These are the first proved cases of tularemia in man to be recorded.

Tularemia is only secondarily a human disease, being primarily a fatal bacteremia of wild rodents. In nature it has thus far been found in the ground squirrel, wild rabbits and hares, wild rats and wild mice. It is transmitted to man by the bite of an intermediate host, by the bite of contaminated animals or by self-inoculation. The intermediate hosts include horse-flies, wood-ticks, bedbugs and fleas. In most of the cases in man, however, the disease is contracted through self-inoculation or contamination of the hands or conjunctival sac with parts of the internal organs or body fluids of the infected animals. Therefore, it is found chiefly in those handling these animals, such as farmers, market men and women, cooks, hunters and laboratory workers. There is no record of the transmission of the disease directly from man to man.

The incubation period varies from one to ten days, with an average of three and one-half days. The onset is sudden and usually consists of headache, nausea, chills and fever, aching bodily pains and sweating. There is enlargement and tenderness of the regional lymph glands. The condition runs a rather prolonged course of two or three weeks with remissions of temperature, recurring chills, sweats, loss of weight, weakness and even prostration. During the first week there is bacteremia. Convalescence is slow, and there is great weakness on exertion.

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* Submitted for publication, May 1, 1929.

* Read before the Omaha and Council Bluffs Ophthalmological and Otolaryngological Society, April 17, 1929.

1. McCoy, G. W., and Chapin, C. W.: Bacterium Tularense the Cause of a Plague-Like Disease of Rodents, Pub. Health Bull., 1912, vol. 53.

Relapses of fever lasting from six to eight days have been reported after eight and ten months, and occasionally there are recurring mild attacks of fever. One attack, however, confers immunity in man.

The diagnosis is made by the agglutination of *Bacterium tularense* by the patient's blood serum. This reaction is absent during the first week, but appears during the second. There is an abrupt rise in titer during the third week. It is thought that the agglutinins never disappear entirely. Martin [2] reported a case in which there was a positive agglutination nineteen years after the original infection. A cross-agglutination also exists with *Bacillus abortus* and *Bacterium melitensis*.

The treatment consists chiefly in the prevention of inoculation or contamination from handling infected animals, for, after the disease is once established, the treatment is purely symptomatic. Even autogenous vaccines have not yet proved satisfactory.

From a careful analysis of 679 reported cases, Dr. Edward Francis [3] concluded that there are four clinical types: (1) ulceroglandular, in which the primary lesion is first a papule and later an ulcer of the skin and is accompanied by enlargement of the regional lymph glands; (2) oculoglandular, in which a conjunctivitis is the primary lesion and there is enlargement of the regional lymph glands; (3) glandular, in which the enlarged regional lymph glands are present but there is no primary lesion, and (4) typhoid, in which there is neither a primary lesion nor glandular enlargements.

I shall now consider the oculoglandular type.

In 1914, Vail [4] first recognized and reported a case of ocular infection as tularemia. This was confirmed by the laboratory work of Wherry and Lamb.[5] His report is a classic description of "squirrel plague conjunctivitis" or conjunctivitis tularensis.

This disease typically presents multiple, small, discrete ulcers of the palpebral conjunctiva with yellow, necrotic plugs and indurated margins. There is marked chemosis and swelling of the lids with a mucoid discharge. The regional lymph glands are swollen and tender. The fever is of the intermittent type, and the convalescence is prolonged. It is to be differentiated from Parinaud's conjunctivitis, conjunctivitis infectiosa necroticans (Pascheff), sporotrichosis, blastomycosis and leptothrix infection (Verhoff). This differentiation, however, is easily made by the blood serum agglutination.

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2. Martin, A.: Tularemia, Southwestern Med. **10**:249, 1926.

3 Francis, Edward: Tularemia in Tice: Practice of Medicine, Hagerstown, Md., W. F. Prior Company, Inc., 1928, vol. 3, p. 663.

4. Vail, D. T.: Bacillus Tularense Infection of the Eye, Ophthal. Rec. **23**:487, 1914.

5. Wherry, W. B., and Lamb, B. H.: Infection of Man with Bacterium Tularense, J. Infect. Dis. **15**:331, 1914.

The second case was reported by Sattler [6] in 1915. Two years later Lamb [7] reported the third case and first recommended an autogenous vaccine, believing that its use had greatly shortened the course of the disease in his case. In 1925, two cases were reported; the fourth by Martin [8] and the fifth by Pfunder.[9] In 1926, twelve more cases were reported,[10] and since then reports have rapidly appeared as the knowledge of the disease has become more widespread, until cases have been reported from every section of the United States except New England. The only foreign report is by Ohara of Japan in 1925.

Francis [3] has compiled thirty-two cases of the oculoglandular type, of which twenty-nine are unilateral and three bilateral. In these the disease was contracted by skinning or dressing rabbits in twenty-one cases, and by handling or crushing ticks or flies with the fingers in three cases. In two cases the source of the infection was uncertain.

To these I wish to add the following two case histories. The first is typical of the oculoglandular type.

REPORT OF CASES

CASE 1.—*History.*—Mrs. T. M., a farmer's wife, aged 29, was first seen on Nov. 13, 1928, complaining that a week previously she had contracted a cold or influenza with chills and high fever from working in cold water during the menstrual period. Since then her right eye had been sore, and there had been some discharge especially in the morning. A more careful history revealed that on the evening of Nov. 4, 1928, she had skinned five rabbits, all of which appeared to be healthy. Forty-eight hours later she became sick with headache, general malaise, chills and fever. She ached all over and could find no comfortable position. Her right eye was sore and red, and there was some discharge.

The next morning she had a temperature of 102 F. and was nauseated but did not vomit. The glands in the right side of her neck were swollen and tender, and she felt some tenderness and swelling under the left arm. The lids of the right eye were stuck shut. On the sixth day she noticed that the index finger of the right hand had begun to swell and a small open sore oozed serum. This persisted until the morning of the examination when there had been some pus.

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6. Sattler, Robert: Bacillus Tularense Conjunctivitis, Arch. Ophth. **44**:265, 1915.

7. Lamb, F. F.: Conjunctivitis Tularensis, with Report of a Case, Ophth. Rec. **26**:221 (May) 1917.

8. Martin, Ancil: Tularemia Infection of Conjunctiva (Case Report), Southwestern Med. **9**:232 and 237 (June) 1925.

9. Pfunder, M. C.: Primary Tularaemia of the Eye, J. A. M. A. **85**:1061 (Oct. 3) 1925.

10. Vail, D. T., Jr.: A Case of B. Tularense Conjunctivitis, Arch. Ophth. **55**:236, 1926. Freese, H. L.; Lake, G. G., and Francis, Edward: Four Cases of Tularaemia (three fatal) with Conjunctivitis, Pub. Health Rep. **41**:369 (Feb. 26) 1926. Flick, J. B.: Tularemia, Ann. Surg. **83**:737, 1926. Martin (footnote 2). Jackson, S. W.: Conjunctivitis Tularensis, J. A. M. A. **87**:123 (Jan. 8) 1926. Morrison, W. R.: Tularemia and Conjunctivitis, J. A. M. A. **87**:607 (Aug. 21) 1926.

On the seventh day a pustule appeared 1 inch (2.54 cm.) below the angle of the jaw on the right side, which opened and discharged pus but which was practically healed two days later. She had been confined to bed for the first four days and after that could stay up only a short time because of weakness.

Examination.—On examination she appeared to be rather ill, although she said she was feeling better that day. Her temperature was 101.6 F., and her pulse rate 106. The anterior cervical glands were enlarged and tender. Moderately enlarged tender glands were palpable in the left axilla. The index finger of the right hand presented a small punched-out ulcer with raised edges in the center of the flexor groove between the distal and middle phalanges.

The left eye was normal. The right eye presented moderate swelling of the lids, mild chemosis and a seromucous discharge. Eversion of the lower lid revealed six discrete round ulcerations of the palpebral conjunctiva which appeared to be punched out and which contained yellowish necrotic plugs. A few were shallow, but some extended through the substantia propria. They were uniform in size, being about 1 mm. in diameter. The surrounding conjunctiva was swollen and congested. The upper lid, bulbar conjunctiva and cornea were not involved. The anterior segment and fundus were normal. Vision was 20/20-1 (missed one letter in 20/20 line).

Treatment and Course.—Smears of the secretion were negative. An agglutination test was reported as positive by Dr. John T. Myers, bacteriologist for the University of Nebraska College of Medicine. Instillations of 1 per cent mercurochrome-220 soluble, corrosive mercuric chloride ointment 1:3,000 and hot applications were prescribed for the eye. Moderate doses of salicylates were given, and a saline bath was advised for the finger.

Five days later, the patient returned complaining that for two days she had had some epigastric pain on deep inspiration; also that for three days there had been more pain and discharge from the right eye, and that there was a tender swelling in front of the right ear. Examination showed the six ulcerations of the lower lid still present, but those in the outer half of the lid showed induration of the margins. In addition, there was a similar small ulcer at the center of the upper lid about 2 mm. from the margin of the lid. Vision was still 20/20-1. The preauricular gland was swollen and hard. The finger showed a loss of the superficial layers of the skin about 2 cm. around the original lesion but appeared to be healing. The patient was advised to continue with the same treatment.

She returned home and has not been seen since. However, her family physician reported that the eye healed in a few days, and that after a rather long convalescence she had fully recovered.

The second case is atypical in that the ocular lesion occurred in a case belonging to the ulceroglandular type.

CASE 2.—*History.*—F. D., a garageman, aged 42, was first seen on Nov. 20, 1928, by my associate, Dr. James C. Tucker, complaining that he had contracted the "grip" about a week before and that three days previous to examination the left eye become red and began to itch.

Careful questioning revealed that ten days before while dressing some rabbits he had scratched the fifth finger on the right hand with the end of a rabbit bone. Four days later, he developed a fever and general malaise which he thought was the grip. The next day he noticed swelling and tenderness of the right axillary glands. The following day the left eye began to feel irritable and sore, and at first there was a little discharge in the morning from both eyes.

Examination.—On examination, the right eye was found to be normal with vision 20/20-2. The left eye presented conjunctival congestion and circumcorneal injection. On the upper inner quadrant of the cornea were several dotlike areas which stained with flourescein, while in the pupillary area there were several thin nebula indicating a previous involvement. A mild iritis was present. Vision was 20/100. There was no involvement of the regional lymph glands. The primary lesion on the distal phalanx of the right little finger showed the typical punched-out ulceration with indurated edges but was clean and healing in. There were no general symptoms except weakness. The agglutination at this time was reported negative by Dr. John T. Myers. However, with blood taken one week later *Bacterium tularense* was promptly agglutinated.

The eye was treated with hot moist applications, atropine, 1 per cent mercurochrome, corrosive mercuric chloride ointment and was kept bandaged. By the fourth day the dots had coalesced, forming a typical dendritic ulcer on the upper inner part and across the cornea below the pupil. The slit lamp revealed slight inflammatory infiltration in the superficial layers of the stroma beneath the epithelial lesion but no keratic precipitates. The healing was uneventful leaving nebula. By the tenth day there was no stain, and vision was 20/50 with correction.

The patient has been seen at intervals since, and there has been no recurrence.

COMMENT

The typical ocular lesion in tularemia is conjunctivitis. In mild cases ulcerations may be absent. However, the classic case presents many small discrete ulcers of the palpebral conjunctiva.

In rare cases, the bulbar conjunctiva is involved. Vail [11] reported a limbal nodule which encroached on the cornea for 2 mm., and Clark [12] described a case in which there was a single ulcer from 4 to 5 mm. in diameter on the bulbar conjunctiva.

Only two references to corneal involvement could be found. Pfunder [9] mentioned that the cornea was "steamy" in his case, and Francis [3] recorded a corneal perforation with prolapse of the iris.

As far as I have been able to determine, my second case is the only one in which a dendritic keratitis occurred during the course of the disease.

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11. Vail (footnote 10, first reference).

12. Clark, C. P.: Atypical Conjunctivitis Tularensis, Am. J. Ophth. **11**:280, 1928.

RECURRENT DERMOID CYST

SUCCESSFUL TREATMENT WITH TRICHLORACETIC ACID *

H. GIFFORD, M.D.
OMAHA

In 1923,[1] I reported five cases of dermoid tumors and two of oil cysts which I scrubbed out with practically pure liquefied trichloracetic acid. My conclusions were that while for well defined, somewhat globular dermoids lying near the surface, this method has no advantage over excision, it is useful in deep-lying tumors, especially when diverticula branch off from the main cavity into the orbit, or, as in one of my cases, when a diverticulum of this type runs for a long distance under the skin of the face. The following case illustrates the efficacy of the method after ordinary measures had been tried without success.

REPORT OF CASE

L. B., aged 27, had had a swelling over the right eye all his life. Within three years he had had three operations. Just what was done could not be made out, but from the fact that a considerable amount of the upper margin of the orbit had been removed, it was evident that there had been some determination to perform a radical operation. There was a somewhat ragged scar about 1½ inches (3.8 cm.) long in the eyebrow, and a parallel scar through the skin just below the brow. The supra-orbital tissues were somewhat edematous and hard; they were painful to touch but not red, with sharp notches in the margin of the bone, especially in the outer half of the orbital margin. I made another cut through the eyebrow, opening into a cavity 1½ inches long by one-half inch (1.2 cm.) broad, full of characteristic dermoid material. The cavity reached into a region which would have been behind the bone if the latter had not been removed. Small diverticula extended backward from the main cavity, especially in the outer half. These were full of granulation tissue and had no characteristic lining. The cavity and obvious ramifications were wiped out and packed with gauze. Two days later, when the gauze was removed, the cavity was found to extend upward for three-fourths of an inch beneath the margin of the incision. All the exposed walls were scrubbed with trichloracetic acid and repacked. Three days later still another pocket was found to extend upward and backward for three-fourths of an inch at the mesial end of the wound. Trichloracetic acid was scrubbed into this, and again into the deeper pockets at the outer end of the cavity. The main body of the cavity was filled with thymol iodide, U. S. P., and the patient was sent back to his physician. The wound closed slowly but completely. About three years later, the patient wrote: "In answer to your question about my operation, it was very successful. Haven't had any trouble since."

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* Submitted for publication, May 13, 1929.

1. Gifford, H.: Treatment of Some forms of Dermoid and Other Cysts with Trichloracetic Acid, Arch. Ophth. **52**:448 (Sept.) 1923.

COMMENT

This case shows the advantage both of treatment with trichloracetic acid and of the plan of temporary packing when there is much oozing and the interior of the cyst has previously been operated on. It is probable that in this case, despite the use of trichloracetic acid, some of the diverticula would have escaped treatment if the operation had been completed in one stage. The great advantage of trichloracetic acid over other agents for destroying the lining membrane of the cysts consists in its combined efficiency and harmlessness. It can be used so freely and repeatedly that all the epidermal tissue is destroyed without danger of going too deep. When a diverticulum is at all deep, it should be filled with the liquefied pure trichloracetic acid by means of a dropper with a long enough tip to reach the bottom of the cavity; then with a suitable swab, preferably on a stiff metal applicator, one can make an application that will be certain to miss none of the epidermal lining of the tube. When a considerable portion of the dermoid wall can be excised easily, it is probably better to remove this before using the acid for the deeper portions. Except in secondary or unusually complicated cases, the wound can be sutured just as if excision alone were practiced.

HYPERTENSIVE RETINAL DISEASE*

LESLIE T. GAGER, M.D.
WASHINGTON, D. C.

In studying the cases of patients with arterial hypertension, a consistent use of the ophthalmoscope during the past decade, and the increase in knowledge during this period of the significance of the changes in the retina which accompany high blood pressure, bring the conviction that no single method of examination gives the physician so much direct information regarding the nature of the process and the patient's outlook and need for treatment as the inspection of the eyegrounds.

Next to the basic requirements of diagnosis, a history which covers particularly the presence or absence of the symptoms of circulatory insufficiency in vital organs, especially the heart, brain and kidney, and a physical examination which gives proper attention to these potential points of weakness, it may be said with assurance, I believe, that the evidence found in the fundus speaks more definitely, even than functional tests, for the progress of hypertension and its benign or malignant character. Indeed, the structural changes in the retinal arteries which hypertension brings about may remain, after the heart has failed and the blood pressure fallen, as the only distinctive sign of the previous condition.

In this paper, after a brief account of the early work in medicine and pathology on which this subject rests, those retinal changes which occur with hypertension and the several types of hypertensive disease with which they are associated will be briefly discussed from the point of view of an internist. And if the physician appears to have borrowed, with a somewhat free hand, a means of diagnostic precision which belongs to a domain and to men of high specialization, it has, I think, by way of compensation, given him an enlarged respect for the science and art of ophthalmology. On the other hand, it is certain that the ophthalmologist, gaining in the breadth of his physiologic interest, from the facts contributed by internal medicine, will lend his aid with greater effectiveness than ever, in the recognition, before serious visual or other ocular disturbances arise, of those first stages of hypertensive disease during which the patient is most amenable to counsel and guidance.

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* From the Department of Medicine, George Washington University School of Medicine.

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That the problems of hypertension deserve attention from all who are concerned with it hardly needs statistical restatement in a day when the predominance of circulatory disease and the major part played therein during middle and late adult life by progressive hypertension have been stressed so frequently.

The advantage to physician and ophthalmologist of an understanding of each other's points of view in respect to systemic and retinal disease finds a happy example in Richard Bright. During the eventful winter of 1814-1815, as noted by Thayer [1] in his biographical essay, the young man, aged 25, was studying in Vienna in the ophthalmological clinics of Rust and Beer. A dozen years later, in the "Reports of Medical Cases," [2] with the famous description of the association of kidney disease and dropsical effusion, and particularly in the summary of his observations in 1836,[3] there is significance in finding that Bright fully realized that he was dealing with a disease common to many organs, and that he had not failed to emphasize the frequent appearance of visual disorders in these patients.

The clinician could go no further than this fact until, in 1851, Helmholtz [4] enriched scientific medicine with an instrument for exploring the fundus of the eye, and described briefly the pathologic changes that were thus revealed. The ophthalmoscope was quickly introduced into the clinic by Traube, and there resulted that description of retinitis acompanying albuminuria which has endured until the present.

From the vantage point of these later days, it has been well said [5] that Bright was writing the final chapter in the history of hypertension and its consequences, though at its beginnings he had shrewdly glanced when, as the cause of the hypertrophy of the heart so frequently found with kidney disease, he set down the possibilities of "an altered quality of the blood" or of resistance to blood flow "in the minute and capillary circulation."

His successors continue to carry on the analysis and differentiation of these factors, and it may be said that to recount the search for the metabolites in the blood as possible agents in producing the long since confirmed peripheral resistance would bring this thread in the story of hypertension down to the present time.

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1. Thayer, W. S.: Richard Bright, Guy's Hosp. Rep. **77**:257, 1927.

2. Bright, R.: Reports of Medical Cases, ed. 4, London, 1827; Hosp. Rep. **1**:338, 1836; Tabular View, ibid. **1**:380, 1836.

3. Bright, R.: Cases and Observations Illustrative of Renal Disease, Guy's

4. Helmholtz, H.: Beschreibung eines Augen-Spiegels zur Untersuchung der Netzhaut im lebenden Auge, Berlin, 1851.

5. Ryle, J. A.: Chronic Bright's Disease Without Albuminuria, Guy's Hosp. Rep. **77**:306, 1927.

It suffices to say that this search for the cause of hypertension precipitated one of the great controversies of medicine. On the one hand were those who believed that persistently high blood pressure is always the result of kidney disease. Here again Traube [6] exerted a lasting influence, when he refused to accept the paradox that hypertrophy of the heart and contraction of the kidney could be due to a common cause, and explained the increased pressure in the aortic system as the result of its overfilling from obstruction to blood flow through the damaged kidneys. In various forms and to varying degrees the nephritic etiology of hypertension has had the support of Romberg and his school,[7] in this country by Janeway [8] and Foster,[9] and Volhard [10] has recently said that he still regards the kidney as the key to the problems of hypertension.

It became evident early, on the other hand, that Bright had described a variety of acute and chronic pathologic processes in the kidney; that many instances of contracted kidney occur without albuminuria and dropsy; that chronic hypertension often exists without diminished renal function or histologic changes in the kidney. In 1872, Gull and Sutton [11] had concluded, in opposition to Johnson, that "kidney disease does not give rise to the vascular changes" which they described in the intima and muscularis of the arterioles throughout the body. And among these morbid changes which they found occurring independently of renal disease they noted the "so-called retinitis albuminurica," with the arteries greatly narrowed and sometimes obliterated, and the familiar picture of retinal degeneration and atrophy.

Finally, as a most important chapter in the story, Mahomed,[12] in 1879, described the occurrence of high blood pressure without structural change either in the vascular system or in the kidneys. Two years later he made another significant observation, namely, that in the nephritis of scarlet fever, the blood pressure may become elevated long before albumin appears in the urine, also stating that treatment of patients with high blood pressure usually averts or cures the kidney disease.

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6. Traube, L.: Gesammelte Beitrage, Berlin, 1856, vol. 2, p. 334.

7. Harpuder, K.: Arteriosklerose, Schrumpfniere und Blutdruck, Deutsches Arch. f. klin. Med. **129**:74, 1919.

8. Janeway, T. C.: Nephritic Hypertension, Harvey Lecture, 1912.

9. Foster, N. B.: The Relations of Hypertension to Cardiorenal Disease, Am. J. M. Sc. **164**:808, 1922.

10. Volhard, F.: Die arterielle Hochdruck, Verhandl. d. Deutsch. Gesellsch. f. inn. Med. **35**:134, 1923.

11. Gull, W. W., and Sutton, H. G.: Chronic Bright's Disease with Contracted Kidney (Arterio-Capillary Fibrosis), Med.-Chir. Tr., London **55**:273, 1872.

12. Mahomed, F. A.: Some of the Clinical Aspects of Chronic Bright's Disease, Guy's Hosp. Rep. **24**:363, 1879; Chronic Bright's Disease Without Albuminuria, ibid. **25**:295, 1880-1881.

In its essentials, the modern statement of the position of hypertension has now been made, for fifty years of intensive work have confirmed and amplified these facts of pathologic physiology. In vascular, that is, arteriolar kidney disease, the high pressure is primary, and the renal changes are secondary and part of a generalized arteriolosclerosis. In nephritis, there is evidence, too, of vascular inflammatory changes throughout the body,[13] of which the lesions in the kidney are only a part and occur subsequent to the rise in blood pressure.

These are facts in view of which it is fair, I think, to say that the renal genesis of hypertension has been abandoned by most students in the case of vascular kidney disease or nephrosclerosis, and by a considerable number now even in nephritis. To the conclusion that hypertension is due to widespread constriction of the arterioles of the body, a functional derangement which leads only secondarily to structural change, ophthalmology, it shall be found, has contributed direct visual evidence, in support of and supplementing the clinical and pathologic observations reviewed.

The changes which the ophthalmoscope reveals in the retina in the course of hypertension may be divided into two main groups: First, there are the alterations which are purely vascular in nature. These occur in three stages: (*1*) spasm of the retinal arterioles, (*2*) arteriolosclerosis and (*3*) hemorrhage. In the second group may be placed the lesions which, in contrast, have an inflammatory character, though often there is a vascular component as well. These changes include: (*1*) edema, retinal and papillary, and (*2*) those "spots," of varying form, color, size and extent, which are called "exudate." Further, there are proliferative, degenerative and atrophic changes which follow in the wake of insufficient vascular supply, hemorrhage and exudation, but these are secondary, and like coincident changes in the choroid, require no separate discussion.

If not always so-called, the branches of the central artery of the retina are to be regarded as arterioles because of their physiologic reactions. In the sense of Ricker,[14] these precapillary segments may be considered as specialized neuromuscular organs, responding to weak or strong stimuli with dilatation, but to the usual moderate stimuli of environment and milieu with constriction. The larger vessels respond little if at all to the same stimuli. Hence it comes about that arteriolosclerosis and arteriosclerosis are in all respects—causation, age incidence, histologic structure, anatomic distribution—two separate entities. In the vessels of the retina, then, is seen not senile or decrescent

13. Kylin, E.: Die Hypertoniekrankheiten, Berlin, 1926.

14. Ricker, Gustav: Sclerose und Hypertonie der Innervierten Arterien, Berlin, 1927.

arterial degeneration, but an arteriolar response to the active process which underlies hypertonus and hypertension.

Spasm is the first phenomenon of hypertensive nature to be found in the retinal arterioles. Since Pal popularized the conception of vascular crises, observations of temporary constriction in the retinal vessels have not infrequently been reported, for example, in the acute severe hypertension of eclampsia or such paroxysms of high pressure as occur with suprarenal tumors. Mylius [15] has reported these spastic or tetanic contractions in eclamptic women, and has observed waves of constriction, resembling peristalsis, passing along the retinal arterioles. The purely functional nature of the condition is shown by the return of normal tonus when the eclampsia is successfully terminated.

But when the elevation of blood pressure remains high, then a few months later there may be found the second stage in this evolution of hypertensive vascular disease. When the hypertension is at a lower level and its progress more gradual, years instead of months may pass before these same anatomic alterations are established. The end-result, as O'Hare [16] and his co-workers have pointed out, is a characteristic arteriolar sclerosis, almost invariably associated with hypertension and rarely occurring with normal blood pressure even when the larger arteries are markedly sclerosed.

Therefore, when a patient comes with the history that the family physician found high blood pressure five years before, but promptly "cured" it, and the ophthalmoscope reveals thin tortuous vessels, with irregular lumina and compression at the venous crossings, the conclusion can safely be drawn that the "cure" was temporary, and that hypertension and its effects are a part of the present diagnostic and therapeutic problem. If the patient has high pressure, but the fundi disclose no disease, it is reasonable to assume that the hypertension is of short duration and the prognosis so much more favorable. If with these well defined vascular changes there is normal or low blood pressure, it is usually evidence of myocardial disease. Finally, if hemorrhage is present in association with this small vessel sclerosis, there is suggested, according to its degree, an exacerbation of a relatively benign process, or the occurrence of hypertension of malignant type.

In the case of C. W., a physician, aged 56, the occurrence of a few retinal hemorrhages ten years ago led to a plan of conservative living, and today his blood pressure is lower, and his general health better, than a decade earlier.

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15. Mylius: Tr. German Ophth. Soc.; abstr., Arch. Ophth. **1**:413 (March) 1929.

16. O'Hare, J. P., and Walker, W. G.: Arteriosclerosis and Hypertension, Arch. Int. Med. **33**:343 (March) 1924. O'Hare, J. P.; Calhoun, A. W., and Altnow, H. O.: Etiology of Chronic Myocarditis, J. A. M. A. **90**:1436 (May 5) 1928.

B. H., a woman, aged 37, was seen because of dizziness and failing vision. The general physical examination disclosed no striking observations. The patient's color was good; the heart was not greatly enlarged. The hemoglobin content was 84 per cent and the red blood cells 4,944,000 per cubic millimeter. Many speciments of urine contained neither albumin nor casts; the specific gravity varied from 1.000 to 1.018, the phthalein output was 50 per cent, the urea nitrogen was 15, creatinine 1.6, and plasma chlorides 450 mg. per hundred cubic centimeters of blood. The blood pressure was 280 systolic and 150 diastolic, and there was a great number of fresh hemorrhages. Neither papilledema nor exudation was marked. This patient died in ten weeks of malignant hypertension, and necropsy showed generalized arteriolosclerosis. In the kidneys, which were normal in size, there was no evidence of inflammation.

In other patients the papilledema and cotton wool exudates have been found more prominent in the picture, as in the descriptions of Keith [17] and his co-workers, but in the case of marked vascular disease, they are apparently not inflammatory in nature. Mr. R., for example, aged 42, had loss of vision, and his ophthalmologist found so much retinal hemorrhage and exudate that the patient was given six months to live. Under careful management of diet and hygiene, the blood pressure was brought down, the hemorrhage was absorbed, vision returned, and the man led an active life, married and worked steadily for seven years. Eventually, cerebral hemorrhage occurred. Except for the arteriolosclerosis, the retinas at this time were normal. Ten days later, a bilateral papilledema developed, and shortly after, Cheyne-Stokes' breathing. Both conditions were relieved by repeated lumbar punctures. There developed during the last two months of life a terminal pyelitis, and fresh exudate appeared in the retinas, but no hemorrhage. A low grade uremia, with twitching and slight convulsions, was present during the few days preceding death, but the chemical examination showed no retention of nitrogen.

Turning now, from this example of a transition, to the purely inflammatory group of retinal lesions, edema, in the absence of arteriolar disease, will be found almost always associated with edema elsewhere in the body. In the case of a man, aged 32, the victim of acute glomerular nephritis, with a complicating pericarditis, there were marked pallor and general anasarca, and retinal edema was the only ophthalmologic sign. The blood pressure was not elevated, and the patient died with uremic convulsions eighteen hours after the consultation.

If the blood pressure becomes elevated, which is the rule if acute nephritis is not successfully dealt with and subacute or chronic nephritis

17. Keith, N. M.; Wagener, H. P., and Kernohan, J. W.: The Syndrome of Malignant Hypertension, Arch. Int. Med. **41**:141 (Feb.) 1928.

develops, there are in the eyeground the soft white or buffy spots and patches, without the arteriolosclerosis, for the development of which time and the functional strain of continued hypertension are required. V. W., a man, aged 46, who had added various infections, especially dental, to the chronic abuse of alcohol, came into the hospital in uremic coma. He was pale and edematous; the blood pressure was 200 systolic and 130 diastolic. The ophthalmoscope showed slight edema, some vascular constriction and irregularity, no hemorrhage, but extensive patches of soft white exudate, which six months later have contracted, in part absorbed, and are much sharper and brighter.

What may be called pure clinical types of hypertensive retinal disease have now been pointed out, in one group of which arteriolar spasm, arteriolar sclerosis and hemorrhage are the stages of development in a progressive vascular condition, while in a second group edema and exudate appear, both often before actual vascular disease can be detected, but the latter never, as a rule, without the presence of hypertension. Much more common than the pure types, of course, in patients who are seen comparatively late in the course of hypertension, are clinical pictures in which exudate and hemorrhage, edema, proliferation of connective tissue and of pigment and degenerative and atrophic changes are so intermingled that interpretation of type is somewhat difficult. In the exudation which accompanied severe vascular disease, it is certain, as Wagener's [18] histologic studies have conclusively shown, that the process is not an inflammatory reaction. These structural details need not be described here, nor will there be discussed the theories of what happens to cause hemorrhage, edema and exudation in the capillary bed distal to the constricted arteriolar segments—whether the phenomena are due to angiospasm and ischemia as Volhard has maintained or to dilatation and hyperemia according to Ricker. Here one is concerned with the facts regarding arteriolar function and structure as altered by hypertension which are affirmed, I believe, by the daily use of the ophthalmoscope in these patients.

CONCLUSIONS

The conclusions which, in my opinion, may be safely based on such observations are as follows:

Hypertension is the expression of an increased tonus in the arterioles of the body, which under suitable conditions can be detected by the ophthalmoscope as vascular spasm.

Under the functional or kinetic strain of increased blood pressure, structural changes occur in the intima and media of these arterioles.

18. Wagener, H. P.: Retinitis of Malignant Hypertension, Tr. Am. Ophth. Soc. **25**:349, 1927.

Partly in the nature of a work hypertrophy, these changes are distinct from those of senile arteriosclerosis, and they are independent of antecedent kidney disease. These lesions also can be observed with the ophthalmoscope and are of practical importance in the diagnosis and prognosis of systemic disease.

Hemorrhage into the retina, associated with arteriolosclerosis, indicates the progress of hypertension and, if extensive, suggests the malign character of the process.

Exudation is a phenomenon almost exclusively associated with hypertension. If it occurs without the presence of vascular sclerosis, a purely inflammatory process, including nephritis, is likely. In the presence of retinal arteriolosclerosis, exudation often is not inflammatory. It is evidence of an advanced stage, or with hemorrhage and edema, of the malignant type, of hypertension.

Clinical experience teaches that the differentiation of the inflammatory lesions is of importance because nephritis and vasculitis often show a remarkable response to treatment, particularly the removal of foci of infection. The early recognition of hypertensive vascular disease likewise is essential to the most effective management of the patient. These things the study of the retina reveals, and an insight into the functional nature and evolution of hypertensive disease is a contribution of ophthalmology to medicine which fosters the optimistic belief that the internist is not struggling with an impregnable problem.

FURTHER VISUAL RESULTS WITH TELESCOPIC GLASSES *

LEO L. MAYER, M.D.
ST. LOUIS

Just two years ago, in a paper read before the Ophthalmic Section of the St. Louis Medical Society and published in the *American Journal of Ophthalmology* in April, 1927, I brought to the attention of the medical profession the use of the Zeiss "distal" lenses or telescopic spectacles in cases in which poor vision maintained. Since the publication of that article, many patients have been referred for trial, and others have been ferreted from the usual office practice. Recently, I asked the superintendent of one of the large hospitals in St. Louis how his uncle was faring with his telescopic lenses. He related a letter from the elderly gentleman extolling with praises this ingenious device and inquiring why the lenses were not better advertised so that many more persons might know of their wonderful possibilities and partake of this miraculous invention. Some of the letters received unsolicited from patients, telling of their experiences, will be quoted.

During the past two years only one article has appeared in the literature on this subject, in both American and foreign journals, and although the present series involves only thirty-five cases tested, I feel that the excellent results obtained make the subject of enough importance to warrant a recapitulation of the extreme usefulness of these lenses.

In an extensive article in the *British Medical Journal* of Sept. 8, 1928, Mr. A. H. Levy made this interesting statement, "Telescopic spectacles are low-power, wide angled Galilean binocular telescope made as compact and light as possible, so that they may be used constantly as spectacles in the ordinary way without serious discomfort or disfigurement." He enumerated the precautions necessary in prescribing and more especially in fitting, but did not give concrete results obtained. However, I wish to call attention to actual results in increase of vision.

All patients were examined with the "distals" which have a magnification of 1.8 times. An improvement has been made in the glass as delivered to and worn by the patient. Instead of the heavy, uncomfortable, easily bent frame of former days, a light, easy shell frame

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* Read before the St. Louis Ophthalmic Society.

is fitted to the contours of the patient's face by the local optician, and this frame is sent to Zeiss in New York where the lens is fitted into a metal collar which in turn is welded to the frame. Thus the glass is more comfortable to the patient and at the same time is not so unusual in appearance, which inconspicuousness is an aid in eliminating some of the self-consciousness of the wearer. That the weight is a factor is shown by the following letter from a patient who received the old style frame:

> I am sending back the glasses that I have tried to use since I have come home. I think that if I could get used to them they would be fine but they are so heavy that I can hardly keep them on. I certainly do appreciate your letting me try them . . . ."

Another patient who had a glass for trial said:

> I have tried these glasses out and regret to advise that I cannot use them with any degree of satisfaction; the range of vision is too small for my eyes. It is truly wonderful how they magnify.

Both of these letters are from out of town patients who, although satisfied when examined at the office, when away from periodic instructions, lost the will to persevere and gave up. My present procedure is not to give these patients a lens for trial, but to have them buy it outright if it improves vision to my satisfaction and the patient is able to afford the cost, for I find that if a person has invested in an article, he is more apt to try to use it.

Another letter from a patient who had had her glasses only ten days said:

> I received my glasses January 23, and am enjoying them very much. I can now read the daily papers, also some in magazines, etc. It is wonderful how well I am able to read in such a short time, and it does not seem to tire my eyes at all. I am writing without the glasses. Don't know if I could focus them just right for writing, I have not tried it. In fact my eyes seem a little stronger than when I saw you. I am so thankful I went to you with my trouble and shall always "sing your praises" to my friends.

This letter was from a woman, aged 75 years, with optic atrophy of long standing and immature cataracts.

To those who have heard or read my previous communication on this subject, there will be some repetition of statements and figures. However, I shall not repeat details concerning the construction of the lens or its method of application, but simply the results obtained.

Four types of eyes call for an examination with the "distals." (*1*) those with poor vision with ordinary lenses, especially inability to read fine print; (*2*) those in which something further in an operative procedure is to be done; (*3*) those in which the patient is so unhealthy or elderly that operative procedure is a decided risk, and (*4*) in cases

in which the patient is to be encouraged with the fact that poor vision is always able to be enhanced by the use of distals.

Cases in this series may be divided as follows, according to the condition of the eye: central choroiditis, 4; uveitis, 2; cataracts, 6; scars of cornea, 2; retinitis pigmentosa, 2; senile macular degeneration, 4; glaucoma, 3; optic atrophy, 3; myopia and choroidal atrophy, 5; chorioretinitis (prenatal), 1; albuminuric retinitis, 1; retinal hemorrhages, 1; circinate retinitis, 1 and detachment of the retina, 1.

The condition in all of these cases was, for the most part, complicated, but by enumerating the types one may better gain an idea of just how difficult are these pathologic changes in the eyes. The majority of patients who are subjects for distals are those who are "at the end of their rope," so to speak, in more ways than one. They have had their eyes treated over prolonged periods by many and various oculists and have usually been informed of the fact that nothing further can be done. Then, too, they are at the end of their financial resources, and as the cost of the lenses is from $50 to $70, there is more than one problem to deal with. I cite the case of a man, aged 28, who had had one eye removed as the result of a dynamite explosion. The remaining eye showed a dense scar of the cornea with the iris plastered up against the back of the cornea and no red reflex of the fundus. His visual acuity was hand motion at 2 feet. After consulting many oculists he arrived at our office, and Dr. Wiener performed an optical iridotomy up and temporally, where the cornea was clearest. After three weeks, he was able to read 20/100 or 6/30 with a +20 diopter sphere, and he could read D1 or Jaeger's test type No. 3 at from 4 to 6 inches with some difficulty. With a distal telescopic element of +20 diopters +2 cylinder axis 105 degrees, his visual acuity was increased to 20/80 or 6/24 only, not an exceedingly great difference. However, with a +8 object side lens added, he read D0.5 Jaeger's test type No. 1, and ordinary newspaper print readily. This patient finds that for the street and for picture shows the ordinary lens is better, because the field of vision is greater. Thus a youth has been restored to economic usefulness, and one more candidate has been withdrawn from a blind institution.

Now I shall give a case from the other extreme of life. A man, S. L. B., aged 83, was told he had everything from cataract to optic atrophy, but no one offered any means of help. Financially well provided for, he was able to have an attendant read to him whenever his wife was occupied. The visual acuity in the right eye was 20/200 or 6/60, and in the left 20/80 or 6/24, which was not improved with any ordinary lens. The fundi showed an area of senile degeneration of the retinas in the macular areas. A telescopic element of ± 0 improved his vision to 20/20 or 6/6, and with a +6 diopter object

side lens his attendant was no longer necessary, for reading D0.5 Jaeger's test type No. 1 and ordinary newspaper print was his normal. This is the patient whom I quoted previously concerning advertising of the distals. Is it any wonder he was enthusiastic?

The next case is that of a man of middle life. C. F., aged 54, had a bilateral optic atrophy resulting from a syphilitic infection. He was treated at the Mayo Clinic with malaria and was greatly improved generally, but his sight remained the same. His visual acuity in the right eye was 20/200 or 6/60 and with + 1.5 diopters it was 20/100 or 6/30; in the left eye, hand motion at 2 feet or 60 cm. with a peripheral field of 10 degrees. The distal lens improved the vision of the right eye from 20/100 or 6/30 to 20/30 or 6/9, and he read D0.5, Jaeger's test type No. 1 and ordinary newspaper type with a No. 8 object side lens over this. So, besides curing the patient of syphilis, practically normal vision was secured.

Every case in which the patient is examined with distal lenses is not, of course, a success.

Mrs. S. O. B. entered Barnes Hospital recently with a history of having been seen by Dr. Dean, eleven years previously, at which time he was in Iowa City. She desired an examination of her eyes, diagnosis and treatment, or operation, if necessary. She was sent to my office after complete physical examination by Dr. Harvey Howard, at the instigation of Dr. James. The visual acuity in both the right and the left eye was 20/200 or 6/60. The media were clear, but the retina of each eye showed a secondary pigmentary degeneration with optic atrophy. The fields were moderately contracted, but there was a large central scotoma of each eye which covered over 5 degrees on the perimeter. With the telescopic element, the vision of either eye could be brought to 20/100 or 6/30, and at times the patient was able to read D2 Jaeger's test type No. 12, but only with great difficulty. In this case it was necessary to put a damper on the enthusiasm of the patient. The vision was improved from 20/200 or 6/60 to 20/100 or 6/30, which meant more to the patient than mere figures would show, but, knowing that the distal lens not only magnifies objects viewed but also increases scotomas, I told her that because of the expensiveness of the lens, together with her meager circumstances, and the slight degree of additional vision, I did not feel that the distal lens would be of value to her. Later, I learned that she had rented my prescription of the telescopic lens from Zeiss for trial.

Another failure was the case of Mrs. M. L., aged 81, with bilateral senile macular degeneration. Her visual acuity was: right eye, with + 5 diopters, 6/200 or 2/60; left eye, with + 1.5 diopters, 20/100 or 6/30. Distal lenses improved the visual acuity of the poorer right eye to 10/200 or 3/60, but that of the better left eye could not be

increased from 20/100 or 6/30. The reason for this was the extremely large central scotoma in the left eye.

While on a vacation in Chicago, I had the privilege of examining a patient for an oculist friend of mine. I was fortunate in being able to have the loan of a distal set from Almer Coe & Company. W. C., aged 53, had had optic atrophy of an unprogressive nature for many years. The visual acuity of the right eye with + 1.5 diopters was increased to 20/150 or 6/45, and the patient was able to read D2 Jaeger's test type No. 12 and medium-sized newspaper print with a + 6 diopter object side lens. His hopeless search had ended with some success.

As a mentioned previously, the distal lens is of use to demonstrate to an apprehensive subject in what way poor vision may be improved. Mr. E. T., aged 48, had an optic atrophy with a central choroidal patch in the right eye which reduced his vision to 20/60 or 6/18. The left eye was myopic, but vision could be brought to normal with an ordinary lens correction. His phobia, always present, was that the left eye might also become seriously impaired. With a telescopic element of —1 diopter, the vision of the poorer right eye was brought to 20/20— or about 6/6, and he read D0.5 and Jaeger's test type No. 1 with a + 6 diopter object side lens over this. The telescopic lens was, of course, not ordered, but the patient's fears had been allayed.

In myopia of high degree in which there is not too great an atrophy of the choroid, the results are indeed startling.

Mrs. K. H., aged 64, was wearing a —22 diopter object lens. She had no perception for light with the right eye, owing to enormous atrophic and pigmented changes in the choroid. With her —22 diopter lens over the left eye, her vision was 20/60 or 6/18. An ordinary lens of —24.5 diopters increased it to 20/40 or 6/12. The distal lens correction increased vision to 20/20 or 6/6. With her ordinary correction she read D1, Jaeger's test type No. 8 at 4 inches. The object side lens over her distal lens made her able to read D0.5, Jaeger's test type No. 1 and smallest newspaper print.

In order to give a more comprehensive idea of the results in this series of thirty-five cases, I have prepared a table which is more or less self-explanatory and will give a panoramic view of the entire group. Snellen's unit in both meters and feet is used, the numerator being omitted when possible.

As will be noticed from the table, the results in a number of cases were unsuccessful; this brings up a point that I want to stress. Each case in which I examine the patient with the distal lens is registered in a book and numbered, so that even though the result is not successful, it comes within the scope of this report.

Results Obtained in Thirty-Five Cases in Which Telescopic Glasses Were Used

Case		Age		Vision	Corrected	Distal	Reading	Diagnosis
1	W. O.	76	OD.	50 or 16	50 or 16	40 or 12	D 0.5	Uveitis
			OS.	.....	.....	.....	.....	Prosthesis
2	H. Y.	45	OD.	200 or 60	200 or 60	.....	.....	Central choroiditis
			OS.	100 or 30	100 or 30	75 or 22	D 0.5	Central choroiditis
3	J. M.	68	OD.	Pl.	.....	.....	.....	Central retinal choroiditis with bilateral cataracts
			OS.	200 or 60	75 or 22	30 or 9	D 1.0	
4	W. W.	28	OD.	.....	.....	.....	.....	Prosthesis
			OS.	H. M.	100 or 30	80 or 24	D 0.5	Scar of cornea, plastic iritis
5	S. B.	83	OD.	200 or 60	.....	.....	.....	Senile macular generation
			....	.....	100 or 30	20 or 6	D 0.5	
6	N. F.	46	OD.	30 or 9	20 or 6	15 or 5	D 0.5	Bilateral immature cataracts
			OS.	30 or 9	30 or 9	15 or 5	D 0.5	
7	C. F.	54	OD.	200 or 60	100 or 30	30 or 9	D 0.5	Optic atrophy, bilateral
			OS.	H. M.	.....	.....	.....	
8	K. L.	75	OD.	No pl.	.....	.....	.....	Optic atrophy with immature cataracts-bilateral
			OS.	100 or 30	100 or 30	30 or 9	D 0.5	
9	W. E.	66	OD.	? Pl.	.....	.....	.....	Complicated cataracts
			OS.	100 or 30	50 or 16	40 or 12	D 0.5	Choroidal hemorrhages
10	W. S.	38	OD.	8-200	8-200	200 or 60	D 1.0	Nystagmus, myopia
			OS.	10-200	10-200	100 or 30	D 0.75	Choroidal atrophy
11	M. A.	81	OD.	300 or 90	200 or 60	50 or 16	D 2.0	Glaucoma-bilateral with contracted fields
			OS.	100 or 30	100 or 30	30 or 9	D 1.0	
12	J. T.	87	OD.	80 or 24	80 or 24	.....	.....	Zonular cataracts with chlosterin deposits
			OS.	40 or 12	40 or 12	30 or 9	D 0.5	
13	L. K.	29	OD.	100 or 30	100 or 30	50 or 16	D 0.75	Choroidal lesions of both macula
			OS.	200 or 60	200 or 60	.....	.....	
14	H. B.	17	OD.	.....	200 or 60	60 or 18	.....	Congenital cataracts with nystagmus-operated
			OS.	.....	200 or 60	40 or 12	D 0.5	
15	F. B.	44	OD.	H. M.	.....	.....	.....	Optic atrophy, bilateral
			OS.	200 or 60	200 or 60	100 or 30	D 1.0	
16	M. B.	67	OD.	100 or 30	100 or 30	50 or 16	.....	Central degeneration of the choroid-bilateral
			OS.	50 or 16	50 or 16	20 or 6	D 0.5	
17	H. P.	19	OD.	10-200	10-200	20 or 6	D 0.5	Retinitis pigmentosa
			OS.	5-200	5-200	.....	.....	
18	J. C.	56	OD.	Pl.	.....	.....	.....	Hemorrhages in retinae (diabetic)
			OS.	75 or 22	75 or 22	30 or 9	D 0.5	
19	T. P.	54	OD.	30 or 9	30 or 9	20 or 6	D 0.5	Chronic uveitis
			OS.	100 or 30	100 or 30	30 or 9	D 0.75	Incipient cataracts
20	N. S.	86	OD.	200 or 60	75 or 22	50 or 16	D 0.5	Pigmentary degeneration of both macula
			OS.	200 or 60	100 or 30	60 or 18	D 0.5	
21	E. K.	64	OD.	3-200	.....	50 or 16	D 0.5	Hemorrhage in macula, right
			OS.	20 or 6	15 or 5	.....	.....	
22	J. L.	62	OD.	3-200	50 or 16	20 or 6	D 0.5	Operated-cataract
			OS.	100 or 30	50 or 16	20 or 6	D 0.5	Central choroiditis
23	W. M.	70	OD.	6-200	6-200	200 or 60	D 0.75	Retinitis circinata, bilateral
			OS.	6-200	6-200	150 or 45	D 0.75	
24	H. S.	74	OD.	5-200	30 or 9	15 or 5	D 0.5	Myopia with senile macular degeneration, bilateral
			OS.	6-200	50 or 16	30 or 9	D 0.75	
25	E. T.	48	OD.	200 or 60	50 or 16	20 or 6	D 0.5	Central choroidal atrophy
			OS.	40 or 12	15 or 5	.....	.....	Myopia
26	M. M.	82	OD.	2-200	5-200	10-200	.....	Myopsic atrophy
			OS.	2-200	5-200	10-200	.....	
27	R. H.	9	OD.	+ 1 foot	7-200	200 or 60	D 1.0	Chorioretinitis of prenatal origin
			OS.	no pl.	.....	.....	.....	
28	J. B.	60	OD.	.....	.....	.....	.....	Anophthalmos
			OS.	10-200	40 or 12	20 or 6	D 0.5	Glaucoma-incipient cataract
29	L. G.	73	OD.	.....	.....	.....	.....	Absolute glaucoma
			OS.	200 or 60	50-16	20 or 6	D 0.5	Glaucoma-incipient cataract
30	S. B.	60	OD.	200 or 60	200 or 60	100 or 30	D 2.0	Pigmentary degeneration of retina
			OS.	200 or 60	200 or 60	100 or 30	D 2.0	
31	D. H.	61	OD.	3-300	.....	.....	.....	Hemorrhage in retina
			OS.	70 or 22	70 or 22	15 or 5	D 0.5	Myopia
32	M. L.	81	OD.	6-200	6-200	10-200	.....	Senile macular degeneration
			OS.	100 or 30	100 or 30	100 or 30	.....	
33	C. N.	72	OD.	100 or 30	100 or 30	40 or 12	D 0.5	Bilateral albuminuric retinitis
			OS.	100 or 30	100 or 30	40 or 12	D 0.5	
34	W. S.	76	OD.	200 or 60	100 or 30	50 or 16	D 0.5	Hemorrhagic retinitis
			OS.	200 or 60	100 or 30	50 or 16	D 0.5	
35	K. H.	64	OD.	no pl.	.....	.....	.....	Myopic degeneration
			OS.	2-200	50 or 16	20 or 6	D 0.5	Myopia

I shall not try to explain the methods of fitting lenses; suffice it to say that only with many trials, constant urging, and an unlimited amount of patience is one able even to attempt this mode of increasing sight. I have made no new discoveries, and I do not wish any one to think that the scientific end has been neglected from any cause, save the fact that I preferred to show practical results, for after all, that is what counts.

THE NEWER FORMS OF OPHTHALMIC LENSES*

ALFRED COWAN, M.D.
PHILADELPHIA

Improvements in the form of ophthalmic lenses have been proposed from time to time ever since the thirteenth century when spectacles are said to have been invented. The first article read before the newly formed Royal Society of England, in 1664, was on the subject of an improvement in "optic glasses." In 1669, Christopher Wren made a hyperbolic lens for a conical cornea. In 1783, a patent was obtained for making achromatic spectacle lenses. Even trifocal lenses were proposed by David Brewster as early as 1810. Nevertheless, it is only lately that improvement in the form of spectacle lenses has received serious consideration.

The first lenses were biconvex, biconcave, planoconvex and planoconcave. Although it is known that meniscus lenses were mentioned by Kepler in 1604, and recommended by Hertel in 1716, and although a machine for grinding toric lenses was built by William Stevens as early as 1854, the flat lens was practically the only form of spectacle lens in use until about twenty-five years ago. At this time, while some double convex and double concave lenses were still being worn, the so-called periscopic lenses (+ or —1.25 diopter base) were most common. Toric lenses were rare, despite the fact that, in 1884, Anton Wagner, of Philadelphia, made the first toric lenses in America.

It may seem strange that improvement in the form of ophthalmic lenses was considered unnecessary for so long a time, while only the very best form of lens was demanded for every other purpose. Continued use of flat lenses after the better forms were known and recommended might be thought due to the refusal of opticians to undertake the commercial risk of manufacturing the more expensive lenses. This was not the reason. From time to time since the middle of the seventeenth century, opticians have been unsuccessful in their efforts to create a demand for concave-convex lenses.

The real reason was that only in comparatively recent times has there been an actual need for the better forms of ophthalmic lenses. Until about twenty-five years ago the standard size of a spectacle lens was what is called one-eye (28 by 37 mm.) and it was not the smallest in use at that time; two-eye lenses (25 by 36 mm.) were fairly com-

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mon, and even three-eye lenses (26 by 35 mm.) were often used. Lenses of such small size hardly required correction; however, spectacle styles changed. Larger lenses became the vogue, and with the larger lenses came the need for improvement in form. Consequently, in the last twenty-five years meniscus, contra-meniscus and toric lenses have become more and more popular until, at the present time, probably 90 per cent of all spectacle lenses are deeply curved.

If it is remarkable that so little was done to improve the form of spectacle lenses for more than five hundred years after their invention, it is no less remarkable that during the last few years several manufacturers have, after a great deal of tedious labor and calculation, created what each claims to be the ideal ophthalmic lens. Each enthusiastic proprietor of a secret, patented or trade-marked lens claims that his is the world's super lens, that it gives perfect vision to the extreme margins, that it is a lens of a single piece of glass that will do what the anastigmat photographic lens does—free from errors of both astigmatism and focal power—or that even bifocals are free from both marginal astigmatism and focal error. They tell ophthalmologists that they should unmask their patients, and that ordinary lenses are like a mask with tiny peep holes. Opticians licensed to sell these lenses find it easy to convince a patient of the superiority of a product so widely advertised. In one instance, a patient who was induced to have his prescription filled in one of these forms was given the impression that his new, trade-marked lenses were different and better than those prescribed by the oculist.

The ophthalmologist owes it to himself to be familiar enough with the basic principles of optics to know that not only are most of the properties claimed for these lenses impossible, but that they are not even necessary in an ophthalmic lens.

From the theoretical standpoint an ophthalmic lens should be free from chromatic aberration, curvature of field, distortion, annoying reflections from the surfaces and astigmatism of oblique pencils. All these errors, however, cannot possibly be corrected in a lens made of a single piece of glass.

Clinically, however, it is known that chromatic aberration, distortion and curvature, in ordinary ophthalmic lenses, are not noticeable enough to cause discomfort. Reflections from the surfaces are sometimes annoying, but can usually be remedied by proper adjustment. The spherical aberration that must be corrected in a photographic lens in order to give a clear image to the edge of the plate does not apply to an ophthalmic lens. Only a very small pencil of light, therefore a small portion of the lens, is used at one time to form an image on the retina. The retinal image is distinctly seen only at the fovea. The only aberration in an ophthalmic lens which need be corrected, therefore, is the astigmatism of oblique eccentric pencils.

Tscherning, Percival and others have shown that the astigmatism of oblique eccentric pencils can be almost entirely eliminated in powers ranging between —20 and +7 diopters by giving the lens a certain coflexure, or front-back combination of curves. These corrected lenses are known as point-focal or wide-angle lenses. For each power there are two forms: one deeply curved, and the other a shallower form. In order to be exact, for a given power and thickness, the combination of front and back surfaces must vary for every distance of the object from the eye, and for each position of the lens with respect to a virtual stop behind. For example, in a lens of certain power and thickness, set at a certain distance from the eye the dimensions of which are known, and with a given distance of the object, the astigmatism of oblique pencils can be almost entirely eliminated within a solid angle of 60 degrees by making the lens of proper shape.

If the lens contains a cylinder, only one principal meridian can be corrected—Percival suggested that it be the horizontal. If the axis of the cylinder is oblique the power in the meridian to be corrected must be known. Again, to be exact, for the same person the lens designed for distant vision will have an entirely different coflexure from the lens designed for the reading distance—in a bifocal the upper and lower portions, having different powers and meant to be used at different distances, must have entirely different front-back surface combinations. In a spherocylindric bifocal, there are four powers and two distances to be computed, only one of which can be corrected.

Strictly speaking, no lens of a single piece of glass can possibly be shaped to eliminate the astigmatism of oblique pencils in all meridians unless it is spherical on both sides and placed at a fixed distance before an eye of known dimensions and for a constant object distance. Even in this case the lens is not corrected for any other aberration. No lens of this kind can be made to eliminate entirely both the error of marginal focal power and the astigmatism of oblique pencils at the same time.

Tscherning's convex lenses, in the shallower forms, from plane to + 6 diopters, all have back surfaces which vary slightly from —8.75 diopters. In the concave lenses the front surfaces decrease evenly from about + 8.75 diopters until in the —20 diopter the front surface is plane. These lenses are calculated for a distant object, and for a rear-stop 25 mm. behind the lens.

Percival, using a different method of calculation and a rear-stop of 27 mm., gave a table of curves for powers ranging from + 14 to — 20 diopters for distant vision, and for powers between + 14 and —15 diopters for reading. The back surfaces in his convex distant lenses vary between —6.50 and —7 diopters. In the reading glasses the back surfaces vary between —4 and —5.50 diopters. The front surfaces of the concave distant lenses gradually decrease from + 6.25 diopters to

a plane in the —20 diopter, and from + 4.25 to —2 diopters in the —15 diopter reading glass.

Southall carefully computed several powers, based on the same specifications as those given by Henker for the Zeiss Punktal lens. The coflexures of the Southall lenses, however, are not the same as those of the Zeiss Punktals.

The Punktal, Percival, Southall and two widely advertised lenses—called A and B—are compared with ordinary meniscus lenses (—6 diopter base curve) in table 1. All these lenses are made of glass having the same index of refraction.

Since the coflexures of no two lenses in table 1 are alike, no more than one can possibly be perfect, even for objects at infinity for which all are computed; each one is wrong for the reading distance. Never-

TABLE 1.—*Comparison of Lenses*

	Given Dioptric Power of Lens	Vertex Dioptric Power of Lens	Dioptric Power of Front Surface	Dioptric Power of Back Surface	Residual Astigmatism, Diopters	Thickness, Mm.
Punktal	+4	+4.14	+11	—7.12	0.01	3.20
Percival	...	+4	+10.56	—6.75	0.0064	2.54
Southall	...	+4	+10.66	—6.89103	0.00006	3 00
A	+4	+4.18	+ 9.87	—5.87	Not given	2.9
B	+4	+4.16	+11.25	—7.37	Not given	3.3
Meniscus	+4	+4.20	+10	—6	0.09	3.00
Punktal	+6	+6.13	+13.50	—7.87	0.03	4.1
Percival	...	+6	+12 641	—7	0.01	3.3
Southall	...	+6	+12.93	—7.38546	0.00022	4.00
A	+6	+6.07	+11.62	—5.87	Not given	3.6
B	+6	+6.20	+12.62	—6.87	Not given	4.2
Meniscus	+6	+6.31	+12	—6	0.15	3.2

theless, any one is just about as good as another for all practical purposes. A lens of this kind must be a compromise at best.

The —6 diopter base meniscus should be compared with the other lenses. This lens, one is told, is like a mask with tiny peep holes and should be discarded for A or B. One should notice the + 6 diopter meniscus in which the residual astigmatism is 0.15 diopter. Any one who has done refraction knows how hard it is for the ordinary patient to accept or reject the addition of a 0.12 diopter cylinder. The astigmatism of 0.15 diopter, however, given in table 1, is for a solid angle of 60 degrees, an angle that no one who can possibly help it will maintain. It is unnatural and uncomfortable. At a distance of 33 cm., an angle of 60 degrees covers a tangent distance of 57 cm., about 22½ inches, and the distance to the object tangent along a 30 degree angle (sec) is more than 38 cm., a difference of nearly 0.50 diopter. For a solid angle of 40 degrees, which is more than enough, the residual astigmatism of the meniscus lens of ordinary strength is negligible.

While the —6 diopter base curve meniscus will answer the purpose of a point-focal lens in the commonly used powers, the curves of the

ordinary toric lenses are too variable. This lens, as it is now made, has a constant + 6 diopter spherical power in the front toric surface. The concave surface, therefore, must change for each spherical power of the lens. For this reason the lens is comparatively cheap; the toric surfaces, being more expensive, vary only in the cylindric components. For example, a prescription calling for: right eye, + 1.00 sphere + 1.00 cylinder, axis 90; left eye, + 2.50 sphere + 0.25 cylinder, axis 90, would have a —5 diopter back surface in the right lens, and a —3.50 diopter back surface in the left lens. Again, in a prescription for: right eye, + 3.00 sphere, and left eye, + 3.00 sphere + 0.25 cylinder, axis 90, in which a sphere is given for one eye and a cylinder for the other, the back surface of the right lens would be —6 diopters if the ordinary meniscus were given, and the back surface of the left lens

TABLE 2.—*Curves for A Lenses*

Given Dioptric Power of Lens	Dioptric Power of Front Surface	Dioptric Power of Back Surface
+0.25	+5.62	—5.37
+0.50	+5.62	—5.12
+0.75	+6.62	—5.87
+1.00	+6.62	—5 62
+1.25	+6.62	—5.37
+1.50	+6.62	—5.12
+1.75	+7.62	—5.87
+2.00	+7.62	—5.62
+2.25	+7.62	—5.37
+2.50	+7.62	—5.12
+2.75	+8.87	—6.12
+3.00	+8.87	—5.87
+3.25	+8.87	—5.62
+3.50	+8.87	—5.37
+3.75	+9.87	—6.12
+4.00	+9.87	—5.87
+4.50	+10.87	—6.37
+5.00	+10.87	—5.87

would be —3 diopters, because the front surface contains a constant + 6 diopter sphere. A constant —6 diopter spherical back surface in the commonly used convex powers would be much better; this, however, would make them more expensive, because it would necessitate a greater variety of toric tools, or would require a larger stock of finished or partially surfaced lenses.

With the objectionable features of the ordinary toric lenses in mind, it might be interesting to study table 2. This table shows the curves of the widely advertised A lenses in powers ranging from + 0.25 to + 5 diopters. It will be seen that the curves of both the front and the back surfaces of these lenses are all given in the unusual 0.12 diopter divisions. Instead of a constant front or back power, there are several constant front curves. They share with the ordinary torics the fault of having discontinuous back surface curves. It is true that this is great in no instance. Nevertheless, for an interval of only 0.25 diopter between + 2.50 and + 2.75, there is a difference of a whole diopter between the back surface powers.

While the designers of these lenses claim that they are free from both astigmatic and focal error for a solid angle of 60 degrees, they do not seem to be so particular about a difference of power at the vertex. Extraordinary curves have been chosen to correct the + 0.25 diopter lens out to the very edge, while the vertex power of the + 5 diopter lens is 0.24 diopter stronger than its given power.

Henker, in his "Introduction to the Theory of Spectacles," offered as an objection to Tscherning's lenses that the thickness and vertex power are not taken into consideration; yet the vertex power is seemingly uncorrected in Punktal lenses between —4.50 and + 3.50 diopters. A lens of this shape has less power at the margin than at the vertex; the designers may have purposely left them a little stronger at the center so that there would be less focal error at the periphery, but it seems hardly likely that they would deliberately correct the margins at the expense of the center.

Although the exaggerated claims of the manufacturers of certain "best form" lenses have been criticized, it should not be inferred that these lenses are not considered good, or do not answer the purpose for which they are made; however, in the ordinary powers and for the practical requirements of an ophthalmic lens, they are no better than the —6 diopter base menisci or torics. In some cases they are not as good. While it is necessary to have lenses as aplanatic as possible in almost every other optical instrument, the eye is so constructed that it is unable to appreciate small errors. Extreme precision, even though it were possible, would be wasted in an ophthalmic lens.

Manufacturers should be frank enough to advertise the limitations as well as the advantages of their lenses. I also believe that the curves of these lenses should be known to the ophthalmologist. A physician should have the same knowledge of the properties of his lenses as he has of the therapeutic action of his drugs.

AMAUROSIS FOLLOWING INGESTION OF ETHYLHYDROCUPREIN

REPORT OF A CASE *

B. Y. ALVIS, M.D.
ST. LOUIS

A case of loss of sight following the therapeutic ingestion of ethylhydrocuprein is reported because of the growing interest in the use of this drug for pneumonia and because so few cases of visual disturbance from its use have been reported. That the drug caused the amaurosis in the following case can hardly be doubted from its resemblance to the classic picture of quinine and ethylhydrocuprein hydrochloride amblyopia so often seen and described.

REPORT OF CASE

Miss V. S., aged 22, had an unmistakable attack of pneumonia on Jan. 2, 1929. On the third day her physician prescribed tablets of ethylhydrocuprein which, according to his direction, were given in doses of 4 grains (0.26 Gm.) every five hours with a glass of milk during the next three days and nights, or until she had taken 58 grains (3.75 Gm.) in all. During the third night she had a high temperature (104 F.) and was somewhat irrational. Her father, acting as nurse, placed cold applications on her forehead with two cloths of different colors. She was able to distinguish between the two cloths until late in the night, when she finally slept. On awakening in the morning, she was unable to distinguish light. Her physician was hastily summoned and, on arriving two hours later, found her entirely blind, with pupils widely dilated and fixed. Dr. Albert Hooss was called for an ocular consultation. His examination, which was made about five hours later, revealed widely dilated pupils, disks very pale with slightly blurred margins, retinal arteries practically obliterated, so narrowly were they contracted; and small and threadlike retinal veins. In the right macula, there was a cherry spot in a moderately edematous retina; no cherry spot appeared in the left macula, but a little to the temporal side there was a round deposit of brownish pigment, apparently old.

The condition was unchanged when I saw her ten days later, except that the patient had gained perception of light. It did not change, as far as could be determined, during the following weeks, when she was removed to the Lutheran Hospital, was operated on for purulent pleurisy and gradually recovered.

At the time of discharge from the hospital, on March 4, she was able to locate the windows and detect a bright light. The pupils responded sluggishly to light. The appearance of the fundus was unchanged, except for the disappearance of the cherry spot in the right macula and gradual clearing of the retinal edema. After her discharge from the hospital, thirteen weeks after the onset of blindness, she was able to count fingers at a distance of 8 feet (243 cm.) with either eye, in a fairly wide field taken roughly.

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* Read before the St. Louis Ophthalmic Society, April 26, 1929.

On April 27, at 4:00 p. m., the pupils were wide and reacted sluggishly to light. The patient said that everything was seen through a purple haze which prohibited the differentiation of colors. Vision in either eye was 20/75. The visual fields were restricted temporally from about 25 to 30 degrees, nasally from 15 to 20, below from 15 to 30, and above 15.

Examination of the right fundus revealed a pale and sharp disk; a few small vessels remained on its face. The lamina cribrosa was faintly visible. The superior nasal veins and artery were empty and were visible as a white thread. Other vessels were very thin, and along the arteries there were white streaks. In the region of the macula there was mottled pigmentation where formerly the cherry spot appeared like a bright red hemorrhage. The pigmented area, however, was much larger and not sharply outlined. In one or two other areas there were small retinal spots of pigment as of old small hemorrhage. In the left fundus, the disk was similar to that in the right. Both the superior and the inferior nasal veins and arteries were empty, and the others were very narrow; along the arteries, there were white streaks. Below and temporally from the macula, a round, circumscribed deposit of black pigment which had been observed on previous examinations was unchanged. The appearance of the fundus was that of extreme optic atrophy with extreme contraction of the retinal vessels; the arteries were almost completely empty.

Treatment.—As soon as the diagnosis of ethylhydrocuprein poisoning was made, i. e., about twelve hours after the onset of amaurosis, nitrites were given in increasing doses. General elimination was instituted, in the form of potassium iodide and pilocarpine for diaphoresis. Hot fomentations were applied to improve the circulation of the eyes. Later, increasing doses of strychnine were administered.

COMMENT

Ethylhydrocuprein, as is generally known, is synthetized from quinine, which is first converted into hydroquinine, then into hydrocuprein, and lastly into ethylhydrocuprein. This is a white powder that is insoluble in water but soluble in alcohol, oils and fats. It is available in the powder or in 2 grain (0.13 Gm.) coated tablets. Exposure to the hydrochloric acid of the stomach converts the base into the soluble hydrochloride. Milk is given with each dose to "lock up" the gastric acid and provide for gradual absorption.

Cross,[1] from whose comprehensive paper on ethylhydrocuprein in pneumonia, the foregoing summary of the chemisty is taken, states that the correct dose, theoretically, is 4 grains (0.3 Gm.) every five hours. It has been shown that ethylhydrocuprein is present in the blood stream in quantity sufficient for pneumococcocidal power six hours after thirteen doses of 4 grains each have been given, while twelve hours after fifteen doses have been given, the serum no longer possesses this power (Baldwin and Rhoades[2]).

1. Cross, F. B.: M. J. & Rec. **125**:271 (Sept. 7) 1927; **126**:354 (Sept. 21) 1927.

2. Baldwin and Rhoades: U. S. Pub. Health Service, Hygienic Lab. Bull. 141, 1925, p. 69.

Visual disturbances from quinine were reported as long ago as 1847. Knapp,[3] in his report of three cases, in 1881, quoted Dr. Baldwin [4] as saying that he had seen cases of blindness, convulsions and death from quinine and that he regarded from 60 to 80 grains (3.9 Gm. to 5.2 Gm.) as a poisonous dose. Roosa seems to have given the first detailed account of quinine amblyopia; this was soon followed by the reports of Michel,[5] Knapp [3] and of numerous other writers of the same period.

Barabasheff [6] administered toxic doses to several healthy human volunteers and produced visual disturbances, even transient amaurosis in one case, proving that the condition arose from the toxic effect of the drug and not from the disease for which it was administered. His patients showed a temporary increase in visual acuity preceding the loss of sight. Many of the patients described the loss of sight as the sensation that one had "turned off the lights."

It was noted by Knapp [3] that relapses occur and are brought about by smaller doses. He further stated that the prognosis is favorable, no known case of permanent blindness being reported; also that "no beneficial mode of treatment seems to have been discovered."

De Schweinitz,[7] in a series of experiments on dogs, found that doses of quinine of from 1 to 4 grains (0.06 to 0.26 Gm.) per pound would produce blindness in from three to fourteen hours. One of the animals remained blind for twenty-nine days. He later [8] showed that by repeating the quinine medication at intervals, total blindness could be maintained. Microscopic study revealed true optic atrophy in some cases associated with thrombosis or embolism of the retinal vessels. The atrophy included optic nerves, chiasm and tracts. He concluded that the original effect of quinine is on the vasocenters, producing constriction of the vessels; that changes in the vessels are finally set up, owing perhaps to endovasculitis, and that thrombosis may occur. He found the ciliary and oculomotor nerves intact, and concluded that the drug has a selective action on the optic and auditory nerves.

Holden,[9] in 1898, by more perfect histologic methods showed degenerative changes in the ganglion cells of the retina even as early as the third day after toxic doses were given. Later, more ganglion cells degenerated, and myelin-like material was deposited in the nerve fiber layer. By the seventeenth day, the Marchi stain showed more degen-

3. Knapp: Arch. Ophth. **10**:220, 1881.

4. Baldwin: Medical Times and Gazette, July 10, 1847, quoted by Knapp (footnote 3).

5. Michel: Arch. Ophth. **10**:214, 1881.

6. Barabasheff: Abstr., Ophth. Rev. **10**:121, 1891.

7. De Schweinitz: Ophth. Rev. **10**:49, 1891.

8. De Schweinitz: Tr. Am. Ophth. Soc. **27**:23, 1891.

9. Holden: Arch. Ophth. **27**:580, 1898.

eration of nerve fibers. In a dog killed on the forty-second day of amblyopia, there was almost an absence of ganglion cells. The degeneration of the myelin sheaths could be traced throughout the tract to the external geniculate body and the pulvinar. The ganglion cells of the external body, of the internal geniculate body and of the anterior quadrigeminal bodies were found to be normal. This animal had had but two toxic doses, and retained fairly good vision until death. Holden concluded that the changes were due to constriction of the vessels, in which he found no pathologic alterations, bringing about lack of nutrition. In one case in which administration of nitrites brought about an early amelioration of symptoms, the same degenerative changes were found histologically as in animals not treated.

Birch-Hirschfeld [10] found the ganglion cells shrunken, changes in the nucleus and the chromatin bodies, with degenerative changes in the rods, cones and granular layers. The vessels were normal. He produced similar changes by cutting off the supply of retinal blood surgically.

Interest in ethylhydrocuprein as a therapeutic agent in treatment for pneumococcic infections was aroused by the paper of Morgenroth and Levy [11] in 1911. From that time until about 1916, many hundred patients with pneumonia were treated with ethylhydrocuprein, mainly as the soluble hydrochloride. Morgenroth, in 1914,[12] called attention to its danger to the optic nerves. Uhthoff [13] reported three clinical cases, two of which came to autopsy, and he gave histologic studies of the posterior portions of the eyes and optic nerves. He called attention to differences in the clinical and microscopic picture between quinine and ethylhydrocuprein amblyopia. In two of his cases there was hyperemia of the disk and blurring of the retina, with some widening of the retinal vessels associated with transient amaurosis lasting for several hours. While the changes in ganglion cells could not be definitely studied owing to postmortem changes, he found Marchi degeneration in the nerve sheaths. The fields in these cases were slightly contracted, but the light sense was greatly diminished. Uhthoff gave a comprehensive summary of the literature up to that time, and concluded that visual disturbances occur in from about 3 to 4 per cent of patients treated with ethylhydrocuprein. He had found about fifty cases in the literature to that time. While none of these patients remained permanently blind, many had prolonged serious consequences, permanent pallor of the papilla, nar-

10. Birch-Hirschfeld: Allg. Wien. med. Ztg. **14**:122, 1900.

11. Morgenroth and Levy: Berl. Klinik, vol. 48, pp. 156 and 191.

12. Morgenroth: Berl. Klinik **51**:1829, 1914.

13. Uhthoff: Klin Monatsbl. f. Augenh. **57**:14, 1916.

rowing of the vessels, contraction of the visual fields and a diminished light sense even in cases of good visual acuity.

It appears that generally the standard dose, as advised by Morgenroth, of 1.2 Gm. (i. e., six doses of 0.2 Gm. each) in twenty-four hours had been exceeded in most patients suffering visual loss but not in all; also that one must reckon a certain idiosyncrasy of individual patients. Uhthoff believed ethylhydrocuprein to be somewhat more toxic to the optic nerve than quinine but not so much so as to contraindicate its use. Morgenroth stated that in normal dogs ethylhydrocuprein seems to have no higher toxic effect on the optic nerve than does quinine.

About 1915, Felix Mendel [14] began the use of ethylhydrocuprein administered with an acid neutralizing compound to prevent its too rapid absorption. For this purpose he gave the drug with milk, which would also provide all necessary nourishment. He reported twelve cases in which the patients were thus treated with five doses of 0.3 Gm. each daily until the fever ceased. One patient showed transient amblyopia lasting for twenty-four hours, after which the treatment was resumed without further ill effect. Baldwin and Rhoades [2] reported, in 1921, the complete absence of dangerous symptoms in a series of patients treated at Bellevue Hospital, the insoluble base being used. Cross,[1] in his series of 126 cases, observed no unfavorable ocular symptoms.

It would seem that in the present case an unusual susceptibility to the drug must have existed. Many cases of transient blindness from large doses of the more soluble chloride have been reported, but in few of these have such lasting and extensive visual disturbances been noted, while in this case, at carefully regulated intervals, small doses were administered with quantities of milk to retard absorption. That it is a case of ethylhydrocuprein poisoning can scarcely be doubted, considering the sudden onset after the administration of considerable doses of the drug, the characteristic ophthalmoscopic picture and the bilateral attack. Arterial embolism of both eyes at the same instant is hardly conceivable.

CONCLUSIONS

1. Amaurosis from ethylhydrocuprein administered internally may occur in severe form, though extremely rarely.

2. Treatment is apparently without influence on the condition.

3. Some permanent disability follows even mild cases of ethylhydrocuprein or quinine poisoning; this varies in amount with the severity of the intoxication.

900 Carleton Bldg.

14. Mendel, F.: München. med. Wchnschr., 1915; Dent. Med. **42**:18, 1916.

Ophthalmologic Review

HETEROCHROMIA IRIDES

F. HERBERT HAESSLER, M.D.
MILWAUKEE

The term heterochromia irides implies no more than a difference in color of the irides of an individual. However, after a superficial acquaintance with the facts, it becomes clear that difference in color produced in one iris by the action of iron or copper, or resulting from atrophy secondary to glaucoma or inflammation, is not part of the phenomenon which constitutes the "heterochromia problem." Heterochromia as such was popularized first by Fuchs, who reported a large series of cases. That his paper made physicians conscious of the problem is emphasized by the fact that complicated heterochromia is frequently called the "Fuchs type," though the phenomenon had been reported earlier. The first recorded observation was made by Lawrence, who in his treatise on "Diseases of the Eye," published in 1843, described two cases.

In 1869, Hutchinson reported three cases of heterochromia. In one, a patient, aged 26, had normal visual acuity in both eyes, the pupil of the paler eye being smaller than that of the darker eye. In the other two patients, aged 35 and 36, the lenses of the paler eyes were opaque.

In 1889, Marcus Gunn reported a case of heterochromia associated with cataract in the lighter eye. The iris of one eye resembled that of the father, and the other that of the mother; the hair and lashes were dark.

In the same year, Sym reported ten cases of heterochromia with various complications affecting the lighter eye. The youngest patient was 16 years old. All but two patients came under observation in the third and fourth decade; most of them had dark hair, but two were fair. The irides were brown and blue in eight cases, and brown and light brown in two. One of the parents had dark eyes, the other light; in one case the father is said to have had heterochromia, and in another both parents had light eyes. The complications noted were cataract, vitreous opacities, choroiditis and glaucoma; the last occurred in three cases.

Malgat published two essays. In his nine cases, eight patients had cataract in the paler eye and all patients were brunettes. The color of

the iris was always congenital, and heredity was not demonstrable. He ascribed it to an intra-uterine disturbance of the anterior ocular segment.

In 1898, Bistis described two cases, one of which was complicated by cataract. He assumed a trophic disturbance to be the cause.

In 1902, Fuchs mentioned that eyes affected with heterochromia may show signs of cyclitis as well as cataract in the paler eye. This observation was made independently by Weill, who published reports of seven cases, in four of which there were lenticular opacities and signs of cyclitis in the paler eye.

FUCHS' DESCRIPTION OF THE CLINICAL MANIFESTATIONS

In 1906, Fuchs published his series of thirty-eight cases. He had already mentioned the condition in the ninth edition of his textbook, and thus established priority over Weill, who described seven cases in 1904. He now, however, discussed the disease on the basis of a large series which included histologic as well as clinical observations. Of his thirty-eight patients, twenty-three were males. In chronic cyclitis with precipitates, he noted the preponderance of females, probably due to the fact that the constitutional diseases which give rise to cyclitis preponderate in females. In heterochromia, on the other hand, such constitutional diseases are usually absent. Heterochromia was observed for the first time in these cases from the second to the seventh decade of the patient's life. As a general rule, it was seen early. More than one half of the patients presented themselves before the end of the second decade. The youngest patient was 13, the oldest 64.

In about one half of the cases one iris was brown and the other blue or gray. In one case both irides were brown though of a different shade, and in all other cases there were two different shades of light color. When one iris was blue and the other gray, it was always the blue one which was involved in cataract or cyclitis. The anatomic difference between a blue and gray iris consists not in the amount of pigment, but in the more delicate structure of the trabeculae in the blue iris. The retinal pigment layer on the posterior surface of the iris is equally developed in the two eyes.

Abelsdorf confirmed this condition in albinotic deaf animals with heterochromia. The difference in the color of the irides was usually observed in earliest childhood, though some patients did not notice it until the vision began to fail in one eye.

Though the pigmentation of the posterior layer of the iris does not determine its color, changes in structure are frequently noted in the lighter heterochromic eye. These defects are visible at the pupillary margin and are believed by Fuchs to be secondary changes. Fuchs did

not note the color of the hair in his early cases, but later found that it is almost always brown or black.

In thirty-three of the thirty-eight cases, the paler eye had a cataractous lens. The opacity usually begins in the posterior cortex, as is often the case in complicated cataract. Fuchs never saw typical star and roset forms, but always irregular dust and spiderweb-like opacities. When ripe, the cataract was bluish-white with visible sectoring. In cases which came to operation he convinced himself that the nucleus too was opaque. Thickening of the capsule was noted only once.

Precipitates on the postcorneal surface are an almost constant accompaniment of heterochromia. They were noted only twenty-nine times in the series, but Fuchs believed that he overlooked them in his earlier observations. They are always fine and white, usually small in amount and limited to the lower part of the cornea. He never observed large lardaceous masses, and never hypopyon. These precipitates remain for years. Occasionally the pupil of the paler eye was dilated, which Fuchs ascribed to changes in the tissue of the iris. Usually the fine radial trabeculae in the pupillary zone of the iris are less strikingly developed or even absent. Even when there is no sign of atrophy of the iris a difference in structure is visible when it is examined under magnification. When both irides are pale the blood vessels are visible, but in the heterochromic eye the vessels are less striking, presumably because of a decreased transparence of the tissue. Pain and ciliary hyperemia are exceptional.

As compared with lesions of the anterior segment, vitreous changes are less commonly observed. However, in cases in which the lens has remained clear or has been removed surgically, fine and somewhat coarse vitreous opacities are seen to occur frequently. In two cases there were ophthalmoscopically visible inflammatory lesions in the periphery of the choroid. There are then two complications of heterochromia: an opacity of the lens and a change which from its clinical manifestations Fuchs called cyclitis. There are four cases in the series in which cyclitis existed without cataract. Does cataract occur without cyclitis? Fuchs noted it in 8 per cent of his cases. He pointed out, however, that in his earlier cases an existing cyclitis may have escaped observation, and that the cyclitis may manifest itself only in vitreous opacities without precipitates. The vitreous opacities could, of course, not be seen. Thus the question of the possibility of cataract without cyclitis is left open. Fuchs next gave a detailed histologic description of pieces of iris excised in five cases and of one pair of eyes taken from a cadaver with heterochromia, but since he himself came to different conclusions after a study of better material, reported more recently, it seems unnecessary to abstract the details.

Theoretically, he pointed out the following possibilities as to the relationship existing between the heterochromia, the cataract and the cyclitis:

A. The pale iris results from the cyclitis.

B. The pale iris is the primary condition and the other changes follow.

a. A pale iris is a fortuitous occurrence (a sport).

b. A pale iris is pathologic. An unknown cause has prevented the iris from developing.

1. Such an eye is a locus minoris resistentiae, and becomes affected with cyclitis and cataract because of its low resistance.

2. The same agent which caused the pallor also produces disease of the eye later; namely, (*a*) the cyclitis as a result of which cataract follows or (*b*) primarily the cyclitis as well as the cataract.

Pallor of the iris as the result of inflammation can occur; in heterochromia the pale iris is most often observed in infancy, while the complications begin later. One could imagine the complications to be the result of the pallor of the iris only if the unpigmented iris affords too little protection to the deeper structures. However, even albinotic eyes are not so affected. Heterochromia certainly does occur as a sport; that is, each eye is entirely normal, but one is dark and the other light. Fuchs cited a family in which one parent and some of the children had brown eyes, the other parent and others of the children pale normal eyes, while one child had one eye like the father and the other like the mother. In another family each of a pair of twins had this type of heterochromia.

The slow development of the iris might explain the interval which exists between the beginning of the heterochromia and the complications. But it is unlikely that the complications are the result of a secondary involvement of a less resistant tissue by an unrelated causative agent, as they are an almost constant accompaniment of heterochromia. What the pathogenic agent is that brings about the pathologic changes in the eye is unknown. I can say only that it has an affinity for the anterior segment of the uvea. It acts early in life, possibly before birth; its action persists for years, but is not intensive and produces no changes in the eye other than heterochromia and its complications.

Fuchs thought it probable that the cataract is a result of the cyclitis rather than an independent result of the same primary etiologic agent. The cataract has the characteristics of complicated cataract: It begins in the posterior cortical layer. The nucleus is opaque even in patients of an age when nuclear sclerosis has begun. Nevertheless, it is striking that so mild a cyclitis here produces cataract almost regularly, while in general mild cyclitis is so rarely accompanied by cataract. He suggested further that possibly the causative agent of heterochromia may

act bilaterally; if this were true, an occasional case of bilateral mild cyclitis with juvenile cataract in pale eyes might be the same disease which is called heterochromia when it occurs unilaterally.

By 1908, interest in this condition had become general among ophthalmologists. Lutz then undertook to study the records of cases observed in Zurich in the ten preceding years, to review the literature and to discuss the problem exhaustively. He reported twenty-three cases in detail, and arranged the pertinent facts in a comparative tabulation. Sixty-one per cent of his patients were males, as they were also in the series of Fuchs. The age at which the difference in color of the eyes was discovered varies. In a large number of cases it was said to have been noted at birth, though as a rule children are born with dark gray eyes, and the permanent color does not develop until the end of the second year. In cases in which the difference in the color of the irides develops some time after birth, it can come about through either aplasia or depigmentation. In two cases the former took place; that is, pigment failed to develop in the paler eye as its mate became darker. In three cases an actual depigmentation was observed. Though some of these observations are open to question, there were nine among the twenty-five cases in which there is no doubt that the difference in the color of the irides was congenital. In no case did a patient complain of pain, redness, blurred vision or other untoward manifestations when there were no complications. The patients came to the physician most frequently in the third and fourth decade, a few in the second decade.

In nineteen of the twenty-three patients Lutz could personally verify the fact that father and mother had different colored eyes, and often the color of one eye was like that of one parent while the fellow eye was like that of the other parent. This might be significant were it not that many more people with homochromic eyes are born of parents whose eyes are different colors than are people with heterochromia. Heredity of heterochromia in the sense that one parent of a heterochromic person had heterochromia did not occur. Eight of the patients were dark-haired and dark-skinned. All were totally heterochromic. Lutz found no case with a partially light iris in which there were complications. He was unable to find any relationship to disease elsewhere in the body. Most of the patients had never had any illness. Among the six uncomplicated cases, three showed evidence of paresis of the cervical sympathetic on the side of the paler eye, and one was associated with hemiatrophia facialis progressiva. Of the complicated cases, only three showed signs of paralysis of the sympathetic. The relatively frequent occurrence of lesions of the sympathetic in cases of heterochromia suggests an atrophic disturbance as its etiology. This view is strengthened by the experiments of Angeluca who noted alopecia and a retardation of growth of bones, teeth, sclera and cornea,

as well as diminution in caliber of the choroidal blood vessels and atrophic spots in the uvea, after extirpation of the superior cervical sympathetic ganglion in young dogs, cats, rabbits and monkeys.

The vision was reduced in the eyes with complications, and in four of the six uncomplicated cases the paler eye accepted a greater convex or a weaker concave lens than the darker fellow eye. Whether it is a characteristic of heterochromia for the paler eye to be retarded in its development from the infantile hypermetropic state to the adult emmetropic or myopic state can be decided only by the collection of larger statistics in the future.

All complicated cases had signs of uveal disease in the form of postcorneal deposits, the characteristics of which were like those noted by Fuchs. Two of the cases in Lutz' series were complicated by glaucoma, which he considered as secondary glaucoma because it occurs in young persons and because glaucoma not infrequently is consecutive to chronic cyclitis. He found no inflammatory lesions in the choroid. In three cases a slight haziness of the margin of the optic disk led him to conclude that optic neuritis existed. Aside from the color and the defects of the posterior layer which were visible at the pupillary margin, the iris was free from changes. Externally, the eyes were completely free from signs of irritation, except in one case in which mild ciliary hyperemia was noted. The course is always long and chronic.

Schlippe, in 1910, offered a histologic description of a piece of iris excised from each of two heterochromic bulbs, and found paucity of pigment and a tremendous increase of nuclei in the iris stroma. He stated that his observations in every way agreed with Fuchs, and he concluded that the heterochromic eye is the result of a chronic uveitis of unknown etiology.

In a second paper Lutz presented data on thirty new cases which corroborated his view previously expressed. He contradicted the view of Fuchs that there are two kinds of heterochromia and that the heterochromia and the cyclitis have a common cause, because of the following reasons:

1. There is a possibility of inheriting heterochromia.

2. Uncomplicated heterochromia is common and not as scarce as Fuchs supposed.

3. There is a disproportion between the degree of pallor of the iris and the severity of complications.

4. A long period elapses between the onset of heterochromia and of cyclitis.

Scalinci considered the entire problem and reported his views and those expressed in the clinical and experimental literature exhaustively. His treatment of the relation of the cervical sympathetic system to

heterochromia is most complete, and he explained the entire symptom complex on the basis of sympathetic paresis. The lack of nervous tone brings about an increased permeability of the blood vessels, which accounts for the postcorneal deposits and for the disturbance of local metabolism which results in cataract. The pallor of the iris is considered a trophic lesion dependent on the nervous disturbance.

In 1917 Fuchs presented a minute histologic study of three whole bulbi. He found that the pigmentation of the iris differs in no way from that of a normal pale iris, from which he concluded that the supposed cyclitis is not responsible for the depigmentation. Fuchs postulated an as yet unknown etiologic agent acting in earliest childhood and possibly before birth. Aside from the paucity of pigment which characterizes heterochromia, he found a slight diffuse infiltration of the uvea with lymphocytes and plasma cells. Whether the nests of epithelioid cells, as noted in Schlippe's case and Fuchs' second case, are part of the picture cannot be decided without further observation.

RELATION OF SYMPATHETIC PARESIS TO HETEROCHROMIA

Von Herrenschwand briefly stated the facts hitherto discovered about heterochromia, and emphasized that an important characteristic of Fuchs' heterochromia, i.e., the complicated form, is the structure of the iris. It differs from a normal pale iris in that it is dull in appearance, the vessels are less clearly visible and the system of trabeculae is flat and less delicately marked. He described fifteen cases illustrating the facts of heterochromia. As a striking contrast to this series, he described in detail thirteen cases of sympathetic-paresis heterochromia which he was able to collect in a short time, once his interest in this form had been aroused. It is usually an accidental condition in people who present themselves for refraction or for the determination of visual acuity for military service. In eleven of the cases there was an obviously enlarged thyroid. The characteristic which induced him to consider this type of heterochromia as an entity distinct from the Fuchs type is the structure of the iris. While in the Fuchs type the iris is similar to a normal pale iris only in its color, in this second group the structure of the paler eye is like that of the darker fellow eye, but the anterior limiting membrane of the iris is absent or greatly retarded in its development. The question arises as to what the paresis of the cervical sympathetic system has to do with this phenomenon, since sympathetic paresis is in most cases not associated with a pale iris.

Mayou reported cases in which an injury at birth resulting in undoubted paresis of the sympathetic was also accompanied by heterochromia. On the other hand, sympathectomy which was formerly done frequently for glaucoma does not seem to have been followed by depig-

mentation of the iris. Only Mendel reported one case in which paling of the iris set in a few months after paralysis of the cervical sympathetic. Experimental work too, on the whole, seems to have given negative results. One positive result is reported by Bistis, who noted depigmentation of the iris which began two days after extirpation of the cervical sympathetic ganglion, and which after sixteen months had progressed to a striking difference in color in the two eyes. Numerous extirpations of the superior cervical sympathetic ganglion and sections of the sympatheticus were performed by Lewinsohn, Hertel, Mohr, Metzger and Wölflin, and in no case did they observe a change in the color of the iris. Francke also stated that Saenger never saw a paling of the iris after sympathetic paralysis produced by any of the numerous injuries which he observed during the war. The assumption seems justified, then, that sympathetic paresis can be effective in this respect only if it precedes the development of the anterior limiting membrane of the iris and its chromatophores.

The observations of Muench are suggestive in this matter, though they have not found general acceptance. He believed that he had shown that in addition to the stroma cells there is a thick layer of small primitive ganglion cells in the anterior limiting membrane of the iris, which he associated with sympathetic nerve-endings. This might make it plausible to expect a failure of development of the anterior layer of the iris when sympathetic fibers are absent at the time of the development of this tissue.

In 1919, Streiff discussed heterochromia at length. By this time it had become a real problem, and though he did use illustrative cases of his own, it was his intention to give an orderly account of the accumulated facts and opinions with their significance rather than to add further examples of facts previously established. He agreed with Scalinci in ascribing the postcorneal deposits to a process other than inflammation of the uvea, though he pointed out that this author is mistaken in believing that vitreous opacities are usually absent. Because of the absence of evidence of inflammation, he favored entirely dropping the term cyclitis from discussions of the problem of heterochromia, and thus he dismissed all theoretical controversy concerning this phase of the problem. Even if one conceded that the precipitates are a manifestation of cyclitis, it need not follow that the cataract is produced by cyclitis. So mild a cyclitis rarely leads to cataract, and in many of the reported cases of heterochromia cataract, postcorneal deposits were absent. There is, then, no reason why a direct connection between heterochromia and cataract cannot be assumed. No case of heterochromia in which entire absence of cataract and postcorneal deposits has been observed by an oculist has later become complicated. How-

ever, in cases in which deposits have been observed, it is extremely rare to have the lens remain clear.

Streiff assembled the reports in the literature on heterochromia, and noted that despite the numerous cases which have been observed, only an occasional one has been studied in which the lighter eye was not on the side of the lesion. However, in view of the slightness of the physical signs, Streiff suggested that there may be merely a bilateral difference in tones, and that this may be merely an associated expression of inherited bilateral asymmetry of which the heterochromia is also a sign but not a cause.

The question of the significance of crossed inheritance has not been stressed in the literature, since it was first postulated by Lutz. Since many cases have been reported in which the patient had one eye like that of the father and the other like that of the mother, it is easy to explain the occurrence of simple, uncomplicated heterochromia as a crossing of eye color of the parents. In complicated heterochromia, he assumed that the normal eye is inherited entirely from one parent, while the lighter eye is inherited chiefly from this parent, but the color of the iris is determined by hereditary determinants (Vererbungs-einheiten) from the other parent, and in the course of development these ambivalent hereditary determinants (Vererbungs-einheiten) are incompatible. From the incompatibility a vasomotor disturbance arises, producing precipitates and cataract. The fact that other congenital anomalies are often associated with heterochromia gives weight to the idea that heterochromia is a problem of heredity. To support his theory, Streiff cited a piece of work by Steiger, who after many years of observation concluded that usually eyes are inherited as a whole, but that a "mosaic inheritance" does occasionally occur. However, precise information regarding asymmetrical inheritance of acquired structures is incomplete.

Streiff accepted Scalinci's view of the significance of the vascular disturbance, and expressed the belief that an associated asymmetry of the sympathetic tone is often a factor in the production of the manifestations of heterochromia. He could not agree with Lutz that Prizbaums systematic breeding of Angora cats for heterochromia in any way illuminates the problem. It merely confuses two unrelated questions.

Streiff assembled a number of examples of sector-like partial heterochromia, and assumed that the darker sector is an example of hyperheterochromia. Complications are not observed in hyperheterochromia, though Streiff saw one woman with dark hair and lashes whose right eye was dark while the left was pale with a hyperchromic sector and the usual heterochromic complications. He interpreted this as a double heterochromia, that is, an eye with heterochromia with a dark sector. He held the heterochromia responsible for the complications.

Streiff saw no objection to accepting the view that the paler color of the one eye is due to hypoplasia of the pigment-bearing stroma cells. It is likely that an eye with hypoplastic tissue is more apt to become defective through vasomotor disturbances, such as Scalinci postulates. He discussed at length the possibility of the occurrence of depigmentation of a normally developed iris with comments on the individual cases in the literature, but he concluded that it must occur rarely if at all.

Glaucoma is an exceedingly rare complication of heterochromia, and Streiff is inclined to ascribe a not unimportant rôle in its genesis to a sympathetic disturbance. In a supplement to this discussion, he commented on von Herrenschwand's study and pointed out that the thinness of the anterior limiting membrane is characteristic of every pale iris.

In a discussion of Bistis' paper, Muench stated that in any iris, even a normally blue one, there is no paucity of the stroma cell net. In the blue iris the pigment is present in just as great a quantity as in the brown iris, but it is colorless. If one uses the staining method of Ranvier or Bielschowsky or even Ehrlich's methylene blue, the cell net is striking. The colorless pigment granules correspond to the interstitial granules in the muscle fibers.

In 1926, Streiff published a description of the peculiar structure of the pupillary zone of the paler iris in a typical case of Fuchs' heterochromia. To this he appended a discussion of the papers published on the subject of heterochromia since his last exhaustive consideration of the literature in 1919. Particularly did he reexamine the evidence pertinent to the view of von Herrenschwand that sympatheticus heterochromia is a distinct entity. He agreed with von Herrenschwand so far as the latter is willing to regard this condition, not as a trophic disturbance resulting from sympathetic paralysis, but as a disturbance in development of the anterior limiting membrane of the iris under the influence of the sympathetic. Instead of speaking of a paresis of the sympathetic he prefers to consider the sympathetic symptoms as the result of asymmetric disposition of the sympathetic. It does not seem to him impossible that the sympathetic of darker races represents a different type from that of lighter races. In the partial crossing of hereditary determinants there may occur heterochromia simplex without sympathetic symptoms as well as with sympathetic symptoms, depending on whether only the iris mesoderm or this structure plus the innervative and muscular structure of lid and pupil is involved. He also believed that depigmentation of the iris after frank paralysis of the cervical sympathetic must be accepted as true. He pointed to the observation of Bistis, in 1925, and to four cases reported by Metzger in which late depigmentation of the iris was observed after traumatic destruction of the cervical sympathetic on the same side.

Kranz agreed with von Herrenschwand, as opposed to Streiff, that the structure of the anterior limiting membrane of the pale heterochromic eye differs from that of a normal pale eye. For a limited group of cases he also accepts von Herrenschwand's theory of a sympathetic disturbance before completion of the development of the iris.

On the basis of a review of the literature, he expressed the opinion that there are four types of heterochromia:

1. Heterochromia simplex.
2. Fuchs' heterochromia.
3. Heterochromia with sympathetic paresis on a hereditary basis.
4. Heterochromia secondary to a sympathetic lesion.

In 1928, Lutz reported further observations and discussed the conclusion to which he has come after many years of interest and thought given to the problem.

In an earlier paper he pointed out three ways in which heterochromia irides could occur: 1. There may be a congenital anomaly. 2. One eye is prevented from becoming pigmented to the same extent as the other. 3. One eye loses its pigment.

The first possibility is reported by many mothers as of certain occurrence. Lutz never saw heterochromia in the first days of life, but he corrected his early assertion that children are usually born with dark gray eyes and that the definite color is not established until the second year. In a foundling home he observed blue, brown and black irides, and therefore felt that the statements of mothers on this point can be given credence. However, he has known of several cases of adults who never noticed their own striking heterochromia, which shows that one cannot base important conclusions concerning the problem of heterochromia on statements of fact as to color of the eye when they have not been verified by a physician interested in the problem. The second possibility has been definitely observed by the author, and the third possibility is supported by three cases recorded in his earlier essay in which the mothers of the patients stated definitely that one eye grew lighter. Nettleship, Shapringer, Berg, Fuchs, von Herrenschwand and Streiff cited cases in which the occurrence of depigmentation can hardly be doubted. Schlodtmann described a boy, the brown color of whose iris had been noted and recorded by a physician five years before a second observation when the pupillary zone was still brown but the ciliary zone was grayish blue.

Lutz collected twenty-four observations to corroborate these statements. In a foundling home where new-born children are deposited on a revolving drum built into the wall of the asylum, particular attention is paid to such details as the color of eyes and hair for future indenti-

fication. Of 224 children, the irides of 24, or approximately 10 per cent, were observed later to have undergone depigmentation, and in 16 children striking changes in the color of the hair were noted. This depigmentation is often complete before the end of the second year, though often not before the onset of puberty. When such depigmentation, presumably due to inheritance and not to disease, is known to be a fact, it becomes easy to believe that it can occur unilaterally.

Lutz' article on unilateral inheritance is a collection of individual cases which seem to show one possibility of this type of inheritance. The most striking example is that of a hermaphrodite bird mentioned by Abderhalden. This bird not only has an ovary on one side and a testicle on the other, but one whole side of its body is male and the other side female.

It is usually assumed that the secondary sexual characteristics are developed as a result of the passage of hormones into the blood stream, but a case like this can be explained only by the assumption of a unilateral inheritance determined by an unequal division of the hereditary substance during the process of building up of the germ plasma. We can then also assume that the heterochromia which results from secondary depigmentation of one iris is ascribable to unilateral inheritance.

There is also evidence of an inheritance of heterochromia as a whole. Przibram could produce it at will in angora cats. In man, direct inheritance from one generation to the other was observed by Calhoun, Becker, Bistis, Kranz, Reinhard and Sym. Fuchs and Streiff observed the condition in twins; De Schweinitz and Schleich, in several members of one family, and Crowther, in the five children of a negro family.

HETEROCHROMIA AS AN ABIOTROPHY

Since his first communication, Lutz has observed thirty-two additional cases. These were part of a clinical material of 15,000 recorded cases, or approximately 0.2 per cent. In six of the eighteen uncomplicated cases, sectored monocular heterochromia was noted, and in two bluish sclera and deeply pigmented lids, giving the impression of a "blue eye" from a distance, the condition which Streiff called hyperheterochromia. There was a smaller cornea in the paler eye in only one case, and in one case there was miosis and ptosis pointing to sympathetic paresis. The complicated cases had corneal deposits and cataract in about the proportion previously observed.

The most striking characteristic of this new series observed in Cuba is the scarcity of sympathetic paresis as compared with the earlier series which had been observed in Switzerland. Possibly it is correlated with the difference in frequency of goiter in the two regions. This contrast is also illustrated by the complete absence of sympathetic paresis in the

series of Francke, whose observations were made on material drawn from the German sea coast, while in his Tyrolean cases he found paresis in a large number.

The fact that some patients assert that the paler eye had become paler from a previously more deeply pigmented state does not cause Lutz to agree with Heine nor Galezowski that the depigmentation is the result of the uveitis. In half of his cases a marked degree of heterochromia occurred without complications, and in 10 per cent of the persons observed with this point in mind, paling of the irides was a physiologic phenomenon. In the absence of evidence of tuberculosis in either series, Lutz is inclined to believe the process to be what is called abiotrophy by Gowers. This is not an actual degeneration of the eye, but rather the inability to compensate regeneratively for the wear and tear on the tissues of the ordinary life process.

The evidence of a tuberculous etiology has not impressed Lutz as conclusive, since he never saw a single case respond to any form of therapy. It is true that Heine attributed all the manifestations to tuberculosis, but Francke contradicted this on the basis of negative reactions to the Ponndorf test. Knapp stated that in his cases the tuberculin reaction was positive, but he did not say whether the reaction was general or focal. Török reported a positive focal reaction to tuberculin in five cases. One patient was afflicted with recurrent vitreous hemorrhage, a manifestation now generally ascribed to tuberculous retinal phlebitis. Cure was reported in the remaining cases, but no one else has ever reported such results. Lutz believed that the entire clinical picture is in no way characteristic of an inflammatory process of bacterial origin, but is rather degenerative. If one considers the clinical picture as previously described by Fuchs, namely, the absence of hyperemia, the absence of a constant order of appearance of the various complications, the occasional absence of corneal deposits, the occurrence of cataract without precipitation and the frequent occurrence of a long-standing, severe inflammatory uveitis without cataract, one can hardly accept an inflammatory origin. The fact that the deposits usually occur at puberty and that the cataract occurs at the end of the fourth decade supports the assumption of the abiotrophic nature of heterochromia, since these are the periods of great metabolic changes. The absence of increased degeneration in the senium cannot be used as a counter-argument, as there is no active metabolism at that time of life to throw a greater load on the poorly energized tissues of the heterochromic eye.

Streiff observed that sympathetic paresis was most frequently associated with the congenital type of heterochromia. He suggested that the unilateral sympathetic paresis represents a disturbance of bilateral symmetry which is in coordination with an asymmetry also manifested in the irides. This makes possible a much more unified view of heterochromia

and makes unnecessary the assumption of several categories which, as a review of the literature shows, cannot be clearly delineated because of the many transitional cases. Even if further investigations should prove the correctness of Heine's and Török's contention, one could assume that the attenuated tuberculosis gained a foothold in the eye because the organ was weakened as a result of unilateral defective inheritance. This would not of course explain the occurrence of cataract without deposits, nor would it explain the unique clinical picture which heterochromia presents.

Though unilateral inheritance can explain all forms of heterochromia, there is one point that still needs elucidation. As pointed out by Lutz himself in his second communication, and corroborated by Herrenschwand, sympathetic heterochromia is usually free from complications. Yet one would expect more complications in these cases because the postulated abiotrophy is so much more extensive, involving as it does not only the eye itself but a large part of the sympathetic system as well. Possibly the higher threshold of stimulation of the sympathetic system brings about a less active metabolism of the region with consequently diminished wear and tear on the structures of the eye.

In conclusion, Lutz stated his belief regarding the problem of heterochromia. He reached the following conclusions after about twenty years of interest in the problem and a personal experience with eighty-seven cases: Heterochromia irides always results from unilateral inheritance, whether present at birth or the consequence of unilateral inhibition of pigmentation or of later depigmentation. Hyperheterochromia, in the sense of Streiff, is the result of a plus development of one side. Of Streiff's hyperheterochromia, there are three variants: (1) heterochromia simplex in which there are no complications because a defective tendency becomes corrected by the development of favorable conditions of metabolism; (2) heterochromia with complications when the tissues are prematurely depleted of the energy to function normally; (3) heterochromia with sympathetic paresis when climatic influence brings about a hyperdevelopment in the region of the sympathetic nerve. The resultant increased threshold of stimulation then retards metabolism and counteracts the abiotrophic process. All three forms are based on a hyperdevelopment resulting from unilateral inheritance. There are transitional cases showing that these are not essentially different kinds of heterochromia, but three variants of one fundamental process.

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News and Notes

EDITED BY DR. GEORGE S. DERBY

UNIVERSITY NEWS

Dr. Joseph L. McCool, Portland, Ore., has been elected to the Chair of Ophthalmology in the Medical Department of the University of California, to succeed Dr. Walter Scott Franklin whose resignation took effect on July. Dr. McCool will move to San Francisco at an early date where he will open an office for private practice.

Dr. Joseph A. White, emeritus professor of ophthalmology, Medical College of Virginia, is the recipient of an honorary LL.D. from Mount St. Mary's College of Emmitsburg, Md.

Exercises for the dedication of the Wilmer Ophthalmological Institute will be held Oct. 15 and 16, 1929, under the auspices of the Johns Hopkins University, the Johns Hopkins Hospital and the William Holland Wilmer Foundation. An appropriate program has been arranged including the following addresses: "The Development of Ophthalmology in Europe," by Hofrat Ernst Fuchs, of the University of Vienna; "Some Contributions and Phases of American Ophthalmology," by Dr. George E. de Schweinitz, of the University of Pennsylvania, and "Color Vision and Its Anomalies," by Sir John Herbert Parsons, of the University of London. Readers are invited to attend.

SOCIETY NEWS

American Ophthalmological Society.—At the annual meeting of the American Ophthalmological Society at Hot Springs, Va., from June 10 to 12, Dr. William C. Posey was made president, Dr. Arnold Knapp president-elect and Dr. Emory Hill secretary.

North Dakota Academy of Ophthalmology and Oto-Laryngology.—At the annual meeting of the North Dakota Academy of Ophthalmology and Oto-Laryngology Society at Fargo on June 6, Dr. George M. Constans, Bismarck, was elected president and Dr. Frederick L. Wicks, Valley City, was reelected secretary. The society will hold its semi-annual meeting at Fargo in January.

Detroit Ophthalmological Club.—Dr. Harry V. Wurdemann, Seattle, recently addressed the Detroit Ophthalmological Club and the Milwaukee Oto-Ophthalmic Society on the medical aspects of aviation.

Toronto Academy of Medicine.—Dr. Luther C. Peter, Philadelphia, recently addressed the ophthalmic section of the Toronto Academy of Medicine on "Methods and Results in Perimetry."

Pacific Coast Oto-Ophthalmological Society.—The seventeenth annual meeting of the Pacific Coast Oto-Ophthalmological Society was held in Salt Lake City, July 1-2. An interesting program of papers was presented. Interest in the meeting was greatly augmented by the presence of the members of the Triological Society, who stopped in Salt Lake City on their way to the meeting of that society in San Francisco. Victoria, B. C., was selected for the place of meeting in 1930.

Kansas City Society of Ophthalmology and Oto-Laryngology.—During the Kansas City Annual Clinical Conference, Kansas City, Mo., Oct. 7 to 11, 1929, there will be a joint meeting with the Kansas City Society of Ophthalmology and Oto-Laryngology, at which Dr. W. C. Finnoff, Denver, will give a demonstration on "Histopathology of Operative Complications in the Eye."

GENERAL NEWS

Dr. Ernst Fuchs Awarded Leslie Dana Medal.—The National Society for the Prevention of Blindness announces that the Leslie Dana gold medal for 1929 will be awarded to Dr. Ernst Fuchs, Vienna, Austria, at the International Ophthalmological Congress in Amsterdam, September 10. This is a departure from the usual custom of considering only Americans for the honor. Dr. Fuchs was formerly professor of ophthalmology at the University of Liège. The announcement says, "He has charted the way for all prevention of blindness work accomplished throughout the world in the last forty-five years." He won the prize at the fifth International Congress of Hygiene at The Hague in 1884 with his essay, "The Causes and Prevention of Blindness."

National Society for the Prevention of Blindness.—William F. Morgan, New York, has been reelected president of the National Society for the Prevention of Blindness, and Dr. Francis Park Lewis, Buffalo, vice president. A conference of all organizations in America engaged in the prevention of blindness will be held in St. Louis, Nov. 11 to 13, 1929.

Tariff on Instruments and Hospital Equipment.—The Senate Committee on Finance has tentatively agreed to recommend to the Senate that the duty on surgical instruments remain as it is under the present tariff act, at 45 per cent ad valorem, and the duty on dental instruments at 35 per cent ad valorem, instead of being increased to 70 and 60 per cent, respectively, as provided by the tariff bill passed by the House of Representatives. The committee has approved, however, the proposed increases in the tariff provided for by the House of Representatives' bill on pharmaceutic, surgical and scientific articles and utensils used in hospitals, laboratories and colleges, wholly or in chief value of glass, from 65 to 85 per cent ad valorem, and in the duty on ophthalmoscopes, corneal microscopes, testing or recording instruments for ophthalmologic purposes, colorimeters, hemacytometers and other instruments of the same class from 45 to 60 per cent ad valorem.

Fellowship in the Mayo Foundation.—Dr. Roynold N. Berke, St. Paul, has been assigned as fellow of the Mayo Foundation, Rochester, Minn., and will major in ophthalmology.

International Trachoma Prize.—The Royal Hungarian minister of public welfare and labor offers a prize of 2,000 Swiss francs for an independent work on the etiology of trachoma which indicates valuable progress in this field. Works that have already been published are eligible for the competition. The jury of award will announce its decision not later than Dec. 31, 1931. Essays should be addressed to Eye Clinic 1, Royal Hungarian Peter Pázmány University, VIII, Mária-Utca 39, Budapest, Hungary.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Physiology

OBSERVATIONS UPON THE VASCULARITY OF THE HUMAN OCCIPITAL LOBE DURING VISUAL ACTIVITY. J. F. FULTON, Brain **51**:310, 1928.

A case is reported of a man with a circumscribed angioma arteriale racemosum of the occipital cortex. Following an unsuccessful attempt to remove the tumor, a bruit could be detected over the operative defect. When the patient was at rest with his eyes closed, the bruit was distant and weak, but when he was requested to read, there was a marked increase in the bruit detectable by both auscultation and electrophonograms. This increase in the intensity of the bruit occurred within from twenty to thirty seconds after the patient began to use his eyes and persisted for nearly a minute after the light was turned off. It did not occur with other types of mental effort such as straining to hear the tick of a distant watch or various forms of olfactory stimulation. The mere reflection of a light into the eyes without the addition of a mental effort to see did not appear to increase the bruit. Further, there was a definite increase in the bruit even when the patient attempted to discern objects in the blind halves of the hemianopic fields.

From this case, it would appear that visual effort is associated with increased vascularization of the occipital cortex.

FRANCIS HEED ADLER.

Color Sense

THE POLYCHROMATIC REFRACTION CURVE AS A GRAPHIC METHOD OF RECORDING THE COLOR SENSE. E. BONWETSCH, Arch. f. Ophth. **122**:98 (May) 1929.

The author used the method of subjective skiascopy or velonoscopy (Trantas-Lindner) with spectral lights as light sources. Owing to the chromatic aberration of the optical system of the eye, every patient showed a different refraction for each spectral light. When these refractions were plotted on the ordinate axis and the wave lengths on the abscissa, polychromatic refraction curves were obtained. The author thought that these curves could be used for the diagnosis of anomalies of the color sense; so he really diagnosed protanomaly and deuteranomaly in some of his patients, although (as he admits) these patients did not show any anomaly when tested with Stillings' and Nagel's charts. The author thought that the anomaloscope would have detected the defective color sense in those cases which he diagnosed as anomalies from his polychromatic refraction curve.

KRONFELD.

Refraction and Accommodation

OPHTHALMIC SCHOOL CLINICS. T. HARRISON BUTLER, Pub. Health **42**:251, 1929.

From sixteen years' experience with school clinics, Butler feels that attention to the eyes of young children is of outstanding importance. Children with ametropia or imbalance of the ocular muscles may suffer from asthenopia, which interferes with their education; and, worse still, through defective monocular or binocular vision, they may acquire an inferiority complex which they are apt to carry through life.

Butler is convinced that the work of an ophthalmic school clinic should be done by an expert ophthalmologist, one who has a sound knowledge of general medicine and of the special diseases of children, and never by an amateur. Only in this way can the interest of the child's visual mechanism be best served.

Butler's school clinic is attended by all school children in Coventry who fail to attain 6/9 acuity with either eye. These preliminary tests are carried out by nurses and teachers in the school, standard conditions of test type and illumination being utilized. Shyness of the little patients is overcome by congregating parents and children in a common clinic and by having the children observe the examination of their schoolmates. The examination in each case consists of manifest refraction without and with glasses that the children may possess, of induction of cycloplegia with three instillations of 1 per cent solution of atropine in each eye at half hour intervals, of careful retinoscopy with a flat mirror in a dim but not dark room, of verification of the retinoscopic examination in the trial case, and finally of examination of the eyes by focal light and of the fundi by the ophthalmoscope.

For the adequate correction of refractive errors of children, Butler emphasizes the need for accurate retinoscopy. The common sources of error in retinoscopy that he points out are:

1. *Incomplete Cycloplegia.*—Whenever 3 drops of 1 per cent solution of atropine fails to give the desired result, a prescription for drops at home is given.

2. *Estimate of Retinoscopy Not Made at Macula.*—An estimate of retinoscopy not made at the macula is a common fault and one that may yield several diopters of error. The remedy is to have the child look at the retinoscope mirror and to check the accuracy of his fixation by observing the position of the image on the cornea.

A prescription for glasses is given after the retinoscopic observations are verified at the trial case. No deductions are made for myopia. In hyperopia, if the error exceeds 8 diopters, 2 diopters are subtracted for cycloplegia; if the error is less than 8 diopters, then 1 diopter is subtracted for cycloplegia. In astigmatism, if the cylinder exceeds 4 diopters, from ½ to 1 diopter is subtracted, while no subtraction is made for cylinders of less than 4 diopters. After the lapse of a month, all children to whom glasses are given are reexamined to check up the fit of the frame, the accuracy of the lens and the visual acuity.

Butler looks on concomitant strabismus as a disorder in which the underlying cause is congenital weakness of fusion, and in which the precipitating cause is anisometropia. He does not have much faith in fusion training with amblyoscope, stereoscope and other devices. To overcome difficulties in fusion, he feels that the best aid is to equalize the visual acuity for each eye through complete occlusion of the fixing eye, "so that no light can enter." Atropine in the fixing eye does not suffice. In his experience, the occluded eye does not suffer in acuity. In this way, he feels that 70 per cent of the amblyopic eyes in children under 8 years of age can be brought to full normal acuity at the rate of one Snellen line a month. Failure to restore acuity to an amblyopic eye may be encountered chiefly in boys, but principally in any child who has passed the age of 10 years.

If strabismus persists, after the utmost has been done to revive the acuity of the squinting eye and after proper glasses have been worn for at least a year, then Butler proceeds with operation. He does not recommend free tenotomy, but always an advancement of the appropriate muscle combined with a recession of its antagonist.

Butler looks on myopia as a hereditary tendency and one likely to progress after debilitating disease. He notes that myopia is more prevalent in mining and agricultural communities where close intermarriage is the rule than it is in cities

where the opposite is true. He points out that the introduction of games into the schools of Sweden brought about a decrease in the amount of myopia in Swedish school children. His practice is to allow a myopic child to continue in school in the normal manner, but to insist that outdoor games supplant music lessons, light reading and other confining interests. He cautions against haste in returning to school of a child who has passed through a debilitating illness, until the child has had the benefit of a long convalescence in the open air with plenty of wholesome food.

J. H. WAITE.

STATISTICAL STUDIES OF THE VARIATION OF REFRACTION. E. TRON, Arch. f. Ophth. **122**:1 (May) 1929.

The author used the ophthalmophacometer of Tscherning to determine the curvature of the cornea, the curvature of the anterior and posterior surfaces of the lens, the depth of the anterior chamber and the thickness of the lens. The total refraction was determined by skiascopy under atropine cycloplegia. Tron measured 55 eyes himself; then he used the material which was published by Awerbach (86 eyes) and that of Zeemann (75 eyes). Altogether the observations on 200 eyes were used for the statistics. The variations of each single optical element were considered separately. They were found to follow the "law of error" of Gauss.

The determination of all the optical components allowed the calculation of the axial length. This was done for the 200 eyes, and the variations of the axial length were thus determined. These variations behaved differently from all the other optical elements. The curve which represented the frequency of all the different axial lengths was irregular and showed marked asymmetry and a high peak. These irregularities disappeared if all the cases with a myopia of 6 or more diopters were eliminated. The cases between plus seven and minus six gave a normal frequency curve for the axial length. That indicated that our refraction material consists of two different types. Type one comprises all cases of "normal" refraction (between plus seven and minus six); type two comprises the cases of high myopia. The refractions which were not emmetropia have been considered as anomalies. Now Tron showed that after elimination of type 2 the frequency curve for all the optical elements separately and for all of them together (the refraction) represented normal frequency curve. Therefore Tron concluded that what are usually called anomalies of refraction are just variants of refraction which are bound to occur. Hyperopia should not be considered as the result of stunted or retarded growth, and myopia should not be thought to be the consequence of excessive close work.

In contradiction to a generally accepted view Tron found that the refractive power of the lens varies just as much as the refractive power of the cornea. Therefore all the calculations of the axial length of the eyeball which considered the refractive power of the lens as a constant factor, were wrong. Tron found the axial length of the hyperopic eye between 19.71 and 23.87 mm., that of the emmetropic eye between 20.46 and 25.46 mm. and that of the myopic eye between 21.62 and 34.77 mm. The other optical elements did not show any definite relations to the total refraction of the eye. In other words, it could not be shown that only a certain small range of corneal refractions was found in hypermetropic eyes and another range in emmetropic eyes. Curvature of the cornea, depth of the anterior chamber, and refractive power of the lens seemed to show the same variability and limits of variation in hyperopia, emmetropia and myopia.

It has been mentioned that certain relations were found between the axial length and the refraction of the eye. That means that certain axial lengths (i.e.

the longer ones) are mostly found in myopia. Furthermore, there are relations between the refractive power of lens plus cornea and the axial length. This correlation is negative, or in other words there was a considerable tendency to combine long axial lengths with low refractive power and vice versa. But this correlation was not strong, so that the absolute refraction was always the result of a combination of a certain axial length with a certain refractive power. In other words, an eye may have just the medium axial length. Then by combination with low, medium or high refractive power the eye becomes hyperopic, emmetropic or myopic.

KRONFELD, Chicago.

DOES SCHOOL OR OCCUPATIONAL MYOPIA DEPEND ON CLOSE WORK, OR IS SOME OTHER FACTOR RESPONSIBLE FOR IT? OSKER HEINONEN, Acta Ophth. **6**:238, 1928.

From his observations of the refractive errors in school children from different social strata, Heinonen noted that there was a slightly higher percentage of myopia among the children of intellectual workers, and that persons with myopia who do not belong to the intellectual group but attain higher education are more myopic than their parents. The peasant teacher who is myopic often has a greater myopic error than his progenitor. The percentage and severity of myopia seems to increase in the higher grades of school life. The author's statistics show that near work in school is not the dominating factor in producing myopia. He believes that the intellectual endowment of the person is intimately bound up with myopia. This probably accounts for the greater percentage of myopia in the children of school teachers than in those of other classes.

A. M. YUDKIN.

Methods of Examination

QUANTITATIVE PERIMETRY; PERIMETRY WITH COLOR INDEX. P. QUADISSANT, Ann. d'ocul. **66**:177 (March) 1929.

According to the usual method, at a given distance and with a given index of dimensions, the usual field is much greater with white than with any color, such as red. Therefore, in order properly to determine visual fields, the white index should be much smaller than the color index. If the same sized object for color and white is used, say, 5 mm., the outside limit for the former will be perceived, but the greatest for white will not be seen.

It is much easier for a patient to perceive or not to perceive a quantitative index than one of color. A color object, going from the periphery toward the center passes through various color phases; for example, red may be seen successively as yellow, orange, light red and dark red.

Certain factors considerably influence the extent of the visual field. Ferree and Rand have demonstrated the effect of the variations of the size of the index and the degree of illumination on the field limits of different colors. According to the author, of primary importance is the quality of the color employed. The saturation of the colors, ordinarily used, is variable but is never total or pure.

It is necessary, therefore, to standardize the methods. The author has attempted to solve the methods by using only artificial illumination filtered by colored screens.

It was noticed that the illumination on the arm which carried the index influenced the result considerably. The field of vision for red was much wider when the ambient illumination was reduced.

Three types of lesions were investigated: (1) compression of the optic fibers by a tumor; (2) tobacco, alcoholic optic neuritis, and (3) multiple sclerosis.

1. With a white index, the fields were practically normal, while with the red index, there was a nasal hemianopia. Small paracentral scotomas for red and white were almost superimposable.

2. This type of lesion caused a small scotoma for white and a larger one for red.

3. Retrobulbar neuritis with central scotomas and contraction of the fields was present.

A comparison of the form and color visual fields should not be attempted until the equalization of sensations has been established. To this end, it is necessary to use only known stimuli of dimensions and light.

In certain diseases of the optic nerves, namely, compression, tobacco or alcoholic neuritis and multiple sclerosis, there is a parallelism between the visual fields for form and for color.

H. W. SCARLETT.

GONIOSCOPY; OPHTHALMOSCOPIC EXAMINATION OF THE IRIS ANGLE. TRANTAS, Arch. d'opht. **45**:617 (Oct.) 1928.

This report is a résumé of the procedures which the author has made use of for twenty-nine years. Four methods are discussed. The first, called simple gonioscopy, consists in the use of the ophthalmoscope with a $+10 - +14$ diopter lens, the angle being examined as obliquely as possible. According to the author's own limited statistics, in almost a fourth of the cases the angle can be seen in this way, especially in young people with deep anterior chambers.

In the second method, pressure with the finger through the lid on the region to be examined is added. By this means, the author has been able to see the angle in 94 per cent of myopic eyes, 82 per cent of emetropic and 55 per cent of hyperopic eyes. These percentages can be increased by the use of eserine and the artificial production of proptosis by retrobulbar injection.

The third method consists in the use of a contact glass. The author states that the picture seen by this means is not so clear, but admits that its use is sometimes unavoidable.

The fourth method consists in the use of the slit-lamp and contact glass. This method is too complicated for practical clinical application and in addition requires close cooperation on the part of the patient.

The author next describes the conditions found in normal eyes. A section is devoted to a description of the canal of Schlemm. He then proceeds to the discussion of disease conditions. He emphasized the importance of combining transillumination with this method. With it, detachment of the ciliary body can be diagnosed.

S. B. MARLOW.

THIRTY YEARS OF NONREFLECTING OPHTHALMOSCOPY. H. SPANUTH, Ztschr. f. Augenh. **67**:221, 1929.

Four methods adaptable to nonreflecting ophthalmoscopy are described by Spanuth. The principle involved in the procedure is that the beam of light used for the examination enters the pupillary area at a different point than that of the observers. The ophthalmoscope which is best suited for this purpose is that in which the source of light and the point of observation are reflected on a plane mirror and then thrown on a concave mirror into separate places on the pupillary area of the patient's eye. The apparatus is arranged so that it can be utilized for binocular observations. It is a modification of the Thorner ophthalmoscope.

A. M. YUDKIN.

Optics and Spectacles

THE FUNCTION OF THE LENS CAPSULE IN THE ACCOMMODATION OF THE EYE. E. F. FINCHAM, Tr. Optical Soc. **40**:9, 1928; Brit. J. Ophth. **13**:259 (May) 1929.

Nearly all the observations were carried out on the lenses of animals. The aim was to record the characteristic contour of the surface of the lens when held by its normal suspensions and when allowed to assume its free shape. The method of making these records was to photograph the image of a target as reflected in the surface. The target was of peculiar design, so that its image produced by a convex spherical surface was square. It was fixed at a constant distance from the surface examined and the camera directed straight downward. The conditions under which the observations were made are given and the following conclusions drawn: 1. The lens surface increases in curvature and assumes the accommodated form when released from its suspensions. 2. The conoidal form of lens surface is found only in those cases in which the anterior capsule has a relatively thin central area surrounded by a zone of greater thickness. 3. The primate lens loses its characteristic accommodated form when the capsule is removed. 4. The amplitude of accommodation is small in those animals which have an anterior capsule of uniform thickness. The abstracter, C. Goulden, says that it seems to him that this paper (together with that published in the same journal, vol. 36, no. 5, by Fincham) establishes the Helmholtz hypothesis beyond doubt.

W. ZENTMAYER.

Experimental Pathology

THE THEORY OF THE INTRA-OCULAR PRESSURE AND ITS RELATIONS TO THE INTRA-OCULAR BLOOD PRESSURE. H. SERR, Arch. f. Ophth. **121**:781 (March) 1929.

The paper represents a criticism of the theory of Dieters, Duke-Elder and Baurmann and contains a few new arguments concerning the question whether the capillary pressure is or is not high enough to account for the intra-ocular pressure.

Dieters, in 1925, advanced the theory that there is no constant stream of fluid going through the normal human eye and that there is perfect equilibrium, which is represented by the formula:
Intra-ocular pressure equals intra-ocular capillary pressure minus the colloidal osmotic pressure of the blood.

In order to prove this, Dieters determined the intra-ocular pressure by tonometry, the colloidal osmotic pressure of the blood by osmometry, and devised a new method for the study of the intra-ocular capillary pressure. That was the main part of Dieters' work. His method made use of the phenomenon of the entoptic visibility of the circulation in the retinal capillaries around the macula. This phenomenon can be seen very easily by every one. The best method to elicit it is to look at the blue sky or through a glass with a certain blue tint. If the patient saw this phenomenon distinctly, then Dieters applied pressure to the lateral wall of the eyeball by using a dynamometer which was much like the instrument Baillart used for his experiments. The instrument was fastened on a stand, and the amount of pressure acting on the eyeball could be measured. Dieters gradually increased the pressure with the instrument until the patient noticed that the entoptic phenomenon stopped. Dieters noted the pressure made by the dynamometer on the eyeball at this moment and then removed the instrument. A few minutes later he prepared the patient for the tonometry and applied the

same pressure with the dynamometer as was found necessary to stop the circulation in the retinal capillaries. When the dynamometer just indicated this pressure, the tension was taken. This tension Dieters considered as the intra-ocular capillary blood pressure. In a large number of cases, Dieters regularly found the difference between the capillary pressure by his method, and the colloidal osmotic pressure of the serum equaled the intra-ocular pressure. The same relation applied to glaucomatous eyes, too. In glaucomatous eyes the capillary pressure was found higher than in normal eyes; therefore, the intra-ocular tension was found abnormally high, too. Dieters acknowledged only one type of stream of fluid in human eyes. That was the case in glaucomatous eyes which were successfully operated on (Elliot's trephine) and showed spontaneous fistulation. In these eyes the capillary pressure was still high, while the intra-ocular pressure was down to normal. Therefore, the difference between the two values led to a positive pressure which forced fluid through the chamber system of the eye and out through the trephine hole. Dieters thought that the abnormally increased capillary pressure was the primary and principal cause of glaucoma.

In his criticism of Dieters' theory, Serr first showed that the method of determining the capillary pressure could not be correct. It is the main principle in physiology of circulation that measurements of the pressure in a certain vessel should be done without changing the conditions that are the subject of measurement. Serr pointed out that by forcing pressure on the eyeball until the entoptic phenomenon stops, the intra-ocular pressure is raised up to about 50 mm. of mercury within two seconds. This certainly means an enormous disturbance of the circulation; the intra-ocular veins were compressed first; the blood was dammed back to the capillaries, and the capillary pressure was increased. It is well known that the slightest touch on a normal eyeball is sufficient to cause a collapse of the retinal veins, so that the veins are most likely compressed at the very beginning of Dieters' experiment. Then Serr contended that Dieters' method, if it really led to values approaching the real capillary pressure, was not so accurate as the author states (within 2 mm. of mercury). Furthermore, the tonometry with the tonometer of Schiötz was not so accurate as Dieters thinks. Owing to the different conditions of the eyes measured with the same instrument, the tonometer gives only relative values. Tonometric results must not be interpreted to mean that the tension of the measured eye really is as high as the tonometer chart indicates. The readings should be understood as the arithmetical means of the limits between which the real tension lies. These limits are from 5 to 8 mm. for the lower weights of the tonometer and from 10 to 15 for the higher weights. Therefore, it is impossible to reach a value within 2 mm. of the real pressure as Dieters contended.

Finally, it was impossible to say whether the increased capillary pressure was the primary change that takes place in the glaucomatous eye or whether it just represented a reaction of the eye to the increased tension. The few observations on eyes which were operated on and in which the tension came down to normal did not prove that the capillary pressure was still increased, which must be postulated if Dieters' theory is right.

Aside from polemics, the school of Leber and most of the German and English physiologists agree that the assumption of an intra-ocular capillary pressure as high as 50 mm. of mercury is not justified. There are no ways of measuring the capillary pressure, but all methods for the determination of the pressure in the intra-ocular arteries indicate a lower pressure than that assumed by Dieters. The final conclusion of Serr's paper is that without the assumption of a constant

stream of fluid going through the eye and without a consideration of the fact that the ways of outflow are more or less impermeable in the glaucomatous eye, the mechanism of glaucoma cannot be explained or understood. KRONFELD.

Lids

TECHNIC OF THE MOTAIS OPERATION FOR PTOSIS. W. W. WEEKS, Am. J. Ophth. **11**:879 (Nov.) 1928.

The method of operating is described in great detail. The technic which tends to secure the successful correction of a congenital ptosis consists in tunneling subconjunctivally, allowing the rectus muscle tendon slip to lie behind the conjunctiva, exposing the rectus tendon by a single horizontal conjunctival incision, making a loop suture in the tendon slip, bringing the tendon slip under the tarsal aponeurosis of the levator, and supporting this tendon slip by a fold of the levator tendon.

W. ZENTMAYER.

Conjunctiva

PARINAUD'S CONJUNCTIVITIS. R. A. TOMASSENS, Am. J. Ophth. **13**:721 (Sept.) 1928.

In a girl, aged 12 years, the conjunctiva was greatly engorged. The upper fold showed granulation with a yellow point of beginning necrosis. Granulations were present in the conjunctiva of the lower lid. There was moderate swelling of the preauricular and cervical glands on the left side. Smears and cultures were negative. Pus from a gland was injected into a guinea-pig. Autopsy was later performed on the animal, and gross lesions typical of tuberculosis were found.

W. ZENTMAYER.

Trachoma

ETIOLOGY OF TRACHOMA. H. STÉPONOWA and N. AZAROWA, Microbiol. J. **8**:180, 1929.

The experiments were started immediately after the appearance of Noguchi's preliminary paper, but, owing to the absence of details of technic in taking material and the methods of preparation of mediums, the isolation of *B. granulosis* was difficult. In only a few instances in a study of a large number of cases was anything resembling *B. granulosis* obtained, though the chromogenous rod described by Noguchi as present in association with *B. granulosis* was frequently encountered.

After the detailed monograph was available, the work became more successful, especially with material from cases of incurable trachoma sent from the Girshman Eye Clinic. In seven of ten cases studied between Nov. 16 and Dec. 6, 1928, the authors succeeded in isolating both *B. granulosis* and the chromogenous rod. Noguchi's technic was followed rigidly, and the cultures were grown in a thermostat at 30 C., since room temperature (15 C.) was too low. A mixed growth was always obtained, banal flora being present as well as the two types of organisms described by Noguchi. The chromogenous rod appeared in from three to four days, *B. granulosis* on the seventh to the ninth day and in two instances on the twelfth day. In one instance a pure culture of *B. granulosis* was obtained on leptospira medium, appearing as a diffuse grayish-white growth. On blood agar two types of *B. granulosis* were secured, a small type, the growth of which appeared on the first day as tiny bright, raised, grayish dots; and a larger type, the growth of which was grayish-yellow and tearlike. The second generation on blood agar appears in forty-eight hours. The yellowish or grayish pigment in the case of

the large type does not appear until the fifth or sixth day, and then in or near the condensation water. Older cultures have a yellow or brownish color. Both types grow well on *Leptospira* medium, but not on plain agar or in broth. Morphologically, the small type is rather a coccobacillus; diploid forms occur; in old cultures larger, involuted forms occur.

The chromogenous rod gives some growth on plain agar as round, tearlike, pigmented colonies (gray, yellow, orange). In broth there is a diffuse opacity, with formation of pigment and much "tear" substance in several days.

The optimum temperature for growth of *B. granulosis* was 30 C. as found by Noguchi. Motile forms were not observed by the authors. The size is from 0.3 to 3 microns.

Four *Macacus rhesus* monkeys were inoculated with the cultures of *B. granulosis* isolated, the inoculations being carried out in accordance with the technic described by Noguchi. Only one animal gave a negative result. In the other three, follicles appeared after from eight to nineteen days. In two of the three animals giving positive results, the uninoculated eye became involved.

The authors feel that they have confirmed the identification of the etiologic agent of trachoma and its pathogenic properties for rhesus monkeys.

S. FLEXNER.

TREATMENT OF TRACHOMA BY THE HIGH FREQUENCY CURRENT. L. RAFAIN, Ann. d'ocul. **166**:306 (April) 1929.

The proper treatment for trachoma should be the destruction of the granulations and their replacement by supple cicatrical tissue, without touching the sound conjunctiva. The high frequency current permits this, its destructive action being easily controlled.

Three methods were employed: (1) diathermal-coagulation; (2) glaring sparkling current, and (3) monopolar fulguration.

In using diathermal-coagulation, the intensity of the current oscillates around 300 milliamperes, while the duration is about one fifth of a second. The intensity of the current is difficult to determine, and it is better to give a feeble intensity over a short duration, than risk a massive coagulation of the lid. This method is efficacious but painful.

The second procedure is delicate as to dosage and painful.

The first two give a severe reaction, consisting of swelling of the lids, redness of the conjunctiva and lacrimation.

The third method, that of fulguration, gives little reaction, is not painful and is just as effective as the other two. It is the method of choice.

The three procedures entirely destroy the granulations without touching the tarsus, providing the application has been made properly.

HUNTER W. SCARLETT.

CHAULMOOGRA OIL THERAPY IN TRACHOMA. J. G. SAMKOWSKI, Ztschr. f. Augenh. **67**:330, 1929.

Samkowski presents the therapeutic value of the use of the chaulmoogra oil as reported from various clinics. In view of the varied results, he treated twenty-two patients with trachoma in various stages of the disease. He noted that massaging of the conjunctiva of the lids and fornix with 2 per cent boric acid salve or petrolatum in trachoma complicated by pannus and ulceration gave just as favorable results as the treatment with chaulmoogra preparations. He believes the result obtained depends on the intensity and extent of the application of the

medicament. He concludes that the preparations derived from *Taraktogenus kurzii* and chaulmoogra oil from *Gynocardia odorata* are not specific for trachoma and have no advantage over the treatments for trachoma ordinarily used in the clinic.

A. M. YUDKIN.

Cornea and Sclera

TATTOOING OF THE CORNEA WITH SILVER NITRATE, GOLD CHLORIDE, AND SILVER CHLORIDE. G. KRAUTBAUER, Klin. Monatsbl. f. Augenh. **80**:66 (Jan.) 1928.

The author has found that the results of chemical tattooing of the cornea may be materially improved by using hydrazine hydrate, 2 per cent, as a reducing agent. Animal experimentation showed that the application of 10 per cent silver nitrate to a suitably prepared portion of the cornea yielded a deep brown sienna when reduced by the hydrazine. A combination of gold chloride and silver nitrate gave a deep black when hydrazine was added, and platinum chloride gave the same with less irritation. The color produced by these various reagents was permanent, and no damage to the cornea resulted. The technic for carrying out the method is given in detail.

FRANCIS HEED ADLER.

STARGARDT'S PROPHYLAXIS OF ULCUS SERPENS WITH ETHYLHYDROCUPREINE. HERWICH RIEGER, Ztschr. f. Augenh. **67**:215, 1929.

Rieger observed that the number of cases of infections following abrasions of the cornea were considerably diminished when a 1 per cent ointment of ethylhydrocupreine was used instead of a 3 per cent boric acid salve. He presents a group of 100 cases and classifies them according to occupation and age. The metal worker seems to be more prone to injury than others, and young people are more often affected than older workers. He believes, therefore, that the use of ethylhydrocupreine salve (Stargardt's prophylaxis) as a preventive in ocular trauma is important from an economic and social standpoint.

A. M. YUDKIN.

CAPILLARY CHANGES IN THE NAIL FURROW ACCOMPANYING KERATITIS PARENCHYMATOSA. GEORGE HALLERBACH, Ztschr. f. Augenh. **67**:306, 1929.

From a study of twenty-three cases of corneal inflammation, Hallerbach concludes that there is no specific alteration of the capillary vasculature in the nail furrow accompanying parenchymatous keratitis. There are, however, some changes in capillary bleeding, in the caliber of the capillaries and in the involutions of the intermediary vessels which are constant in these cases. He believes that these pathologic phenomena are not syphilitic but are produced by disturbances in the endocrine system. He assumes that the success achieved by Schnaudigel's administration of iodine therapy in keratitis parenchymatosa substantiates his contention of endocrine influence in this disease.

A. M. YUDKIN.

Lens

DEMONSTRATION OF CALCIFICATION OF THE LENS IN VIVO. HANS MEVES, Ztschr. f. Augenh. **68**:30, 1929.

Meves reports a case in which a complicated cataract was examined in vivo by the roentgen ray. The roentgenogram demonstrated a well defined shadow in the lens. The outer phase of the density was heavier in outline than the nuclear part. The process was localized in the lens tissue. Circumstances demanded an

enucleation of the eye. A chemical analysis of the chalky white lenticular substance confirmed the diagnosis of calcification of the lens. A histologic study revealed an old perforation of the sclera adjacent to the shadowed area. The iris was adherent to the lens capsule, and the ciliary body was also distorted. The hematoxylin-eosin stain revealed the entrance of the opacity at the place of injury and also other minor areas of beginning calcification.

A. M. YUDKIN.

Vitreous

THE CILIARY BODY IN CASES OF LUXATION OF THE LENS. E. FUCHS, Arch. f. Ophth. **122**:86 (May) 1929.

Fuchs described an anomaly of the ciliary body which he found in eight cases of his own material. In these cases the ciliary processes were pointing backward, toward the orbiculus ciliaris and were close to each other, just as though they had been pulled down by some kind of traction on the part of the zonular fibers which are inserted on the pars plana. This condition was found in eyes which showed a congenital ectopia of the lens, only in the lower, nasal and temporal parts of the ciliary body. The upper part of the ciliary body was normal. Zeemann found the same condition in a case of congenital ectopia and explained it by the traction of the meridional zonular fibers.

Fuchs believed that this condition was due to the change of the position of the lens. During the course of a congenital ectopia, the lens usually slides down toward the lower ciliary body. The lower margin of the lens itself pushes the ciliary processes down. From there, the lens goes on sliding backward with its lower margin. The lower zonular fibers are still intact; they still connect the ciliary processes with the lens. Therefore the ciliary processes must move with the lens on its way backward. The same changes take place in the lateral parts of the ciliary body, but not in the upper part where the zonular fibers are destroyed in an early stage of the process. The dislocation and distraction of the ciliary processes may lead to slight pain and irritation, but probably not to real inflammation.

KRONFELD.

Glaucoma

GLAUCOSAN IN GLAUCOMA. D. K. PISCHEL, Am. J. Ophth. **11**:705 (Sept.) 1928.

The substance was employed in simple glaucoma. The series was small and the cases were not long under observation. The conclusions drawn were that in the cases observed levoglaucosan, an epinephrine substitute, always brought the tension down; in some cases after its use, miotics again became more effective. There seems to be some possibility of its causing a rise of tension; so the patient should always be under observation.

W. ZENTMAYER.

THE SYMPTOMATOLOGY OF GLAUCOMA AND THE PROBLEM OF PATHOGENESIS. A. MAGITOT, Ann. d'ocul. **166**:356 (May) 1929.

The first question that confronts one in dealing with glaucoma is hypertension. To the clinicians they mean one and the same thing. They should not be confounded, however, as glaucoma is a syndrome (Demicheri), and hypertension is one of the elements. The more glaucoma is analyzed the more two symptoms come to the fore, hypertension and optic atrophy.

In each patient the ocular tension goes through a daily cycle. It goes down during the day, commences to mount during the night, and reaches its maximum

during the early morning, resembling the temperature curve. In two persons the cycles of the ocular tensions may oscillate differently, but the early morning tension is always the highest, while the general curve is parallel to the pressure curve of the humeral artery.

The hardness of the eye not only is variable in each person, but may also be so in each crisis of the same subject. According to Gilbert it is impossible to make a classification of glaucoma from the hardness of the eye, for there are white eyes with extreme hardness, and there are inflamed eyes the tension of which is not over 35 mm.

According to many, glaucoma can exist without hypertension and, on the other hand, hypertension can be present without glaucoma. Increased tension and optic atrophy do not necessarily go hand in hand, nor does hypertension necessarily mean disturbance of vision. Equally true, there is no parallelism between hypertension and irritative symptoms.

The classification of glaucoma as given by Elschnig is as follows: (1) simple hypertension without affecting the optic nerve; (2) hypertension associate with excavation, and amaurosis excavation, termed degenerative glaucoma, and (3) those comprising prodromal, acute and chronic glaucomas.

After analyzing the clinical and anatomic facts, the conclusion is reached that the origin of all glaucoma rests in a vascular condition.

No symptom is more in accord with hypertension than corneal edema, but the degree of hardness does not regulate it. The same can be said of pain in glaucoma. Some cases of comparatively low tension are accompanied by severe pain, while others with high tension have little or no pain.

The depth of the anterior chamber varies, sometimes being deep, and again shallow. This depends on the swelling neither of the vitreous nor of the lens.

The pupil may be contracted, dilated, or any position in between, or it may be fixed. The condition may be temporary, or it may return to normal with great difficulty. Anterior synechiae when present are the result of hypertension and not the cause.

Hypertension is not considered due to mydriasis, although some think it plays a certain rôle.

The author draws the following conclusions: 1. There is no parallelism between the degree of hypertension and optic atrophy.

2. There is no correlation between the hardness of the globe and the painful phenomena, the irritative signs, mydriasis and the depth of the anterior chamber.

HUNTER SCARLETT.

Retina; Optic Nerve

DISCIFORM MACULAR DEGENERATION. B. CUSHMAN, Am. J. Ophth. **11**:720 (Sept.) 1928.

A man, aged 69, presented a history of trauma to the right eye with permanent reduction of vision. Vision in the left eye had been failing rapidly for six months. In the right eye, the mass was prominent and presented areas of scattered pigment. Fine retinal vessels extended over it. In the left eye the mass was flat and showed areas of pigment, new vessels and hemorrhages. In both eyes there was angiosclerosis. Photographs of the fundi by von der Heydt illustrate the condition.

W. ZENTMAYER.

NOTES ON A PEDIGREE OF AMAUROTIC FAMILY IDIOCY (TAY-SACHS' DISEASE). D. D. STENHOUSE STEWART, Brit. M. J. **1**:101 (Jan. 19) 1929.

The author comments on the rarity of references to this condition during the past twenty-eight years as compared to their plentiful appearance in the first

twenty years succeeding its first description by Waren Tay in 1881. He then reports a case of this disease occurring in the second generation of the classical R. family, which was recorded by Kingdon, of Nottingham, in volume 80 of the "Transactions of the Medico-Chirurgical Society." Of twelve children, six died of this complaint, and in two, extensive postmortem examinations were made by Risien Russell. Of the remaining six, the eldest daughter is unmarried. In the family of the elder son, there is one living child, one pregnancy terminated in a "mole," and another child died of meningitis. The patient whose case is reported was the second child of the next daughter, and died in convulsions. A boy born to the same mother is now 7 years of age and well. One other sibling has a healthy daughter aged 2½ years. The author raises the question whether or not the succession is solely through the distaff line. He asks whether matrimony should be advised against and also for suggestions as to the prenatal and postnatal treatment in any further pregnancies.

A. KNAPP.

PARTIAL EDEMA OF THE PAPILLA OF THE OPTIC NERVE WITH SIMULTANEOUS PARTIAL ATROPHY; REPORT OF AN INTERESTING CASE. G. GRAF and J. MIKULINSKA, Arch. d'ophth. **45**:288 (May) 1928.

The authors point out that a tumor in the anterior part of the orbit results in venous stasis and choking of the disk, whereas a tumor in the posterior part produces atrophy of the optic nerve. According to Byers, when the point of penetration of the vessels into the nerve is affected, the choking of the disk increases. Some cases have been reported in which atrophy has been followed by choking. The reverse is much more common. In the literature available to them, the authors were able to find reports of only two other cases in which edema and atrophy were present at the same time.

They next describe in detail the case of a girl, aged 18 years, who had proptosis of the left eye which moved freely except for a slight paresis of the internus. Vision was reduced to 1/60, and the lower half of the field was almost completely lost. Ophthalmoscopic examination showed the upper half of the disk to be white and flat, whereas the lower half was congested and swollen about 3 diopters. At operation the nature of the growth made it impossible to save the eyeball, and exenteration of the orbit was done. Pathologic examination showed the tumor to be a myxosarcoma which probably arose from the sheath of the nerve. For a distance of 9 mm. behind the globe, the nerve was not involved.

The authors then attempt to correlate the ophthalmoscopic with the anatomic observations. The microscopic picture indicates that the atrophy followed edema caused by pressure on the nerve at the point of origin of the tumor. They discuss at some length the probable mechanism by which this was accomplished.

S. B. MARLOW.

CHOKED DISK AND SEROUS EPENDYMITIS. F. TERRIEN, G. RENARD and M. A. DOLLFUS, Arch. d'ophth. **45**:288 (May) 1928.

The authors describe two cases in detail. The symptomatology of each was similar. Intense occipital headache, unrelieved by analgesics and aggravated by physical effort, paroxysmal in character and of varying duration, was the chief complaint. Vomiting of a migrainous type accompanied the exacerbations of headache.

The authors particularly emphasize the visual disturbance, which is rarely lacking in the presence of increased intracranial pressure. It consists in a clouding of vision, a fogging, which obscures vision for a few seconds. It is sudden in onset but of brief duration, and recurs frequently during the day and is perhaps

related to the increase in the hypertension. Whatever its pathologic change, it should lead immediately to the diagnosis of increased intracranial pressure.

Choking of the disk, which was present in the authors' patients, is a most important symptom and needs no emphasis. In their cases, it seemed to come on shortly after the establishment of the symptoms. They watched its increase and decrease with the rise and fall of the intracranial pressure. One patient had an abducens paralysis which was transitory. No other disturbances were present in their cases.

In view of the disturbance of vision, no delay in energetic treatment is permissible. Although the loss of vision does not remain complete, nevertheless the patient does not entirely recover from it. Early diagnosis is therefore most important, and for this lumbar puncture is necessary. The spinal manometer indicated increased pressure in both cases reported by the authors.

The increase in pressure having been established, its cause must next be determined. The authors then discuss the differential diagnosis of specific meningitis, tuberculous meningitis, abscess of the brain and tumor of the brain in relation to the two cases reported.

In regard to treatment, they advocate the energetic administration of mercury and arsenic, even when syphilis can be ruled out, as long as the visual acuity and the field of vision remain normal. To this, lumbar puncture with the removal of small amounts of spinal fluid is added.

If the hypertension does not recede, if visual acuity diminishes and the field of vision contracts, operation is imperative in order to preserve vision.

Finally, these patients must be watched over a period of years because of the possibility of recurrence.

S. B. MARLOW.

Toxic Amblyopia

RHINOGENIC RETROBULBAR NEURITIS. O. THIES, Arch. f. Ophth. **122**:75 (May) 1929.

During the last two years the subject of the rhinogenous etiology of retrobulbar neuritis was discussed at three great congresses in Germany. It was found that there were many ophthalmologists who strongly believed in the existence of this condition. On the other hand, some of the most outstanding clinicians denied altogether the occurrence of retrobulbar neuritis caused by inflammatory conditions of the sinuses. These men contended that it never occurred, that a patient with a retrobulbar neuritis consulted the otolaryngologist first and that the otolaryngologists never saw eye complications in their sinus cases. It was found by Scheerer that in about 55 per cent of all the cases of retrobulbar neuritis multiple sclerosis was present or became manifest soon after the eye symptoms started out. The opponents of the rhinogenous theory contended that most of the "rhinogenous" cases would have shown symptoms of multiple sclerosis if the patients had been seen five years after the retrobulbar neuritis commenced. Hippel said at the last meeting of the German Ophthalmological Society that even in cases in which the rhinologist found something there was no sufficient proof that the sinus condition had anything to do with the retrobulbar neuritis. The results of surgical procedures on the sinuses, which were reported by many authors, were in Hippel's opinion misinterpreted and overestimated.

Thies reported two typical cases and presented all the facts which strongly indicated the relations between sinus conditions and the orbital part of the optic nerve. One of the reasons that the optic nerve was affected in only a few of the sinus cases was that special anatomic conditions must be present. These conditions

were described by Herzog (1928). In some of these cases the sheaths of the optic nerve were in close contact with the medullary spaces which perforated the lateral wall of the posterior ethmoidal cells. In some other cases it looked as though the optic nerve was going through the ethmoidal cells. These special anatomic conditions were necessary for the development of eye complications. Thies pointed out the necessity of having excellent x-ray pictures. Without an x-ray picture the diagnosis of rhinogenous retrobulbar neuritis could not be made. The results of surgical treatment of the sinuses were always successful; there was always a certain amount of actual and permanent improvement of vision, and no harm was done to the patient. The author thought that those who underestimated the importance of a rhinogenous retrobulbar neuritis were likely to make the mistake of letting an eye become blind just because there were indefinite symptoms which suggested the presence of multiple sclerosis, although the surgical treatment of the sinus disease would have offered a possibility of cure.

KRONFELD, Chicago.

Neurology

A CASE OF RIGHT HOMONYMOUS HEMIANOPIA. NASR FARID, Brit. J. Ophth. **13**:2 (Feb.) 1929.

When first seen, a man, aged 65, could see fingers at a distance of 20 cm. (8 in.) with the right eye; vision in the left eye was 6/9. The fields showed right homonymous hemianopia, with preservation of fixation. In both eyes there were small areas of chorioretinitis. The patient was arteriosclerotic and was given to the excessive use of tobacco and coffee. The diagnosis made by Professor Fuchs was a venous hemorrhage into the tracts on their way up to the occipital lobes. Other symptoms present were presumably due to multiple foci of the same nature affecting the pyramidal tracts and other parts of the central nervous system. The Babinski reflex was absent on the right side. When the patient was examined two years later, the hemianopia had almost disappeared.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

FRACTURE OF THE OPTIC CANAL. O. BARKAN and H. BARKAN, Am. J. Ophth. **11**:767 (Oct.) 1928.

The authors point out that Berlin in 1869 established the true pathology of blindness following blows on the frontal region and reviewed Holder's work in which he showed that in 60 per cent of fractures involving the base of the skull the lesion extended into the foramen opticum. The loss of vision is usually due to a consequent hemorrhage into the sheath or to actual laceration of the nerve within the canal, or to both. In some cases pressure on an anterior clinoid process may be the cause. The paper contains the case reports and field charts of five of the authors' series of twenty-two cases. There is usually a history of blunt injury, often relatively slight, to the outer portion of the superior orbital margin. If the patient is conscious, he notices immediate loss of vision. Either the immediate, almost complete, loss of vision rapidly improves, in which case the cause must have been pressure of blood within the sheath, or, after initial improvement, a permanent defect in the field remains, in which case there must have been in addition a tear in the nerve; or amaurosis remains as the result of complete severance of the nerve. Pallor due to descending atrophy may be observed from the second week on. A sector defect in the field of vision extending to and including fixation is sufficiently characteristic to be almost pathognomonic. These sector

defects are almost typically downward and outward. Where the nerve is torn, the light reaction of the pupil remains in abeyance in spite of the improvement in vision. Theoretically, this is clinical proof of the localization of the pupillomotor fibers in the macular bundle. In these cases central vision will not be restored. The authors suggested that if an early diagnosis is made, operation should be urged in the hope that early removal of the pressure on the nerve may restore vision in some cases in which it would otherwise be permanently lost.

W. ZENTMAYER.

INTRA-ORBITAL MICROGUMMA. F. LARICCHIA, Ann. di ottal. e clin. ocul. **57**:198 (March) 1929.

A woman, aged 21, gave a history of swelling of the upper lid for the past four months with proptosis occurring a few days before her first visit. A mass could be felt in the upper part of the orbit, and the movements of the globe were limited, especially upward. The first Wassermann reaction was negative, but in spite of this the patient was given arsphenamine. The swelling increased and keratitis developed from exposure. A piece of the tumor was removed which showed on section marked infiltration with round cells, especially around the vessels, and inflammatory hyperplasia. The growth seemed to arise in the sheath of the levator muscle and was considered characteristic of a late syphilitic lesion. A second Wassermann reaction proved positive, and further antisyphilitic treatment resulted in the complete disappearance of the swelling and the restoration of normal ocular movements. The author states his belief that many so-called pseudo-tumors of the orbit are probably of syphilitic origin. A bibliography and photomicrographs are included.

S. GIFFORD.

THE DIAGNOSIS OF INFLAMMATORY EXOPHTHALMOS ACCOMPANYING ACUTE INTRA-ORBITAL MICROGUMMA. F. LARICCHIA, Ann. di ottal. e clin. ocul. **57**:198

Inflammatory exophthalmos may originate from (1) infection in the eyeball or (2) in Tenon's capsule, (3) extension from infection in the soft tissues of the face, (4) orbital osteitis, (5) cavernous sinusitis and (6) the sinuses of the nose. The pathologic condition produced by these foci of infection may be (1) a collateral inflammatory orbital edema, (2) subperiosteal abscess, (3) orbital phlegmon and thrombophlebitis or (4) inflammation of Tenon's capsule. Intraocular inflammations produce the same local symptomatology as new growths. The former is accompanied by a varied amount of edema of the orbital tissue so that a clearcut diagnosis is difficult.

The author describes eight cases of exophthalmos following acute inflammatory diseases of the adnexa which he allowed to heal spontaneously. He notes that this lesion in other clinics is interfered with too frequently by surgical intervention. His experience shows that in acute infectious processes producing edema and proptosis operation should not be performed immediately, whereas in acute exacerbations of chronic sinusitis operation should be performed at once, particularly when the nasal examination does not reveal definite local disturbance. An acute sinusitis may be differentiated from a chronic condition by the history of the case, by direct examination of nasal tissue and by the x-rays.

Subperiosteal abscess is found accompanying periostitis and osteomyelitis of the bony orbital socket and is usually confined to the orbital margin. Syphilitic and tuberculous disturbances of the bone are somewhat unusual. They are slow in developing, in contrast to the acute infectious process. Subperiosteal abscess must be differentiated from an orbital involvement, for the method of treatment is some-

what different. At first both conditions are alike, for the edema masks the true lateral displacement of the eye noted in the former. True paresis cannot definitely be elicited, and the visual acuity is often disturbed by accumulated toxic substance in the acute cases. The presence of pupillary dilatation, paralysis of accommodation and corneal anesthesia would indicate a ciliary nerve paralysis and hence an intra-orbital disturbance.

In intra-orbital inflammation there may be thrombophlebitis of the orbital veins or a diffuse abscess-forming phlegmon in the orbital fat. Yet a sharp determination cannot readily be made of these manifestations, for orbital phlegmon may accompany either acute infections or growths in the orbital socket.

It is easy to diagnose proptosis that is produced from infection of the soft tissues of the face. As a rule, these cases are fatal. Operation on the lacrimal sac may lead to exophthalmos, but this is readily diagnosed.

Erysipelas of the face leads to many complications besides proptosis. The most important is a necrotizing abscess of the lids. This lesion calls for surgical intervention. An incision of the abscess on the lid often gives considerable relief to the sufferer.

A. M. YUDKIN.

General Disease

OCULAR CHANGES IN POSTMALARIAL ANEMIA. V. SABA, Ann. di ottal. e clin. ocul. **57**:213 (March) 1929.

The author gives his observations in fourteen cases of severe, often fatal, anemia occurring in the course of malaria. The changes in the fundus were the same as those in severe anemia from any cause, consisting in marked pallor of the fundus, edema of the retina, dilatation of the veins with constriction of the arteries and hemorrhages, which were present in thirteen of fourteen cases. The hemorrhages often showed the surrounding grayish-white zone which sometimes has been considered characteristic of leukemia. The red color of the macula often greatly resembled the cherry spot seen in thrombosis of the central artery. A bibliography is included.

S. GIFFORD.

THE OCULAR COMPLICATIONS OF INTESTINAL INFECTIONS CAUSED BY SPECIFIC ORGANISMS. G. WORMS and GABRIEL SOURDILLE, Arch. d'ophth. **45**:737 (Dec.) 1928.

The authors first call attention to the importance of including the gastrointestinal tract in the search for possible etiologic factors in those diseases of the eye likely to be due to foci of infection.

Under the heading of the colon bacillus group, they first report two cases of iridocyclitis in which improvement occurred while the patient was under a dietary regimen, although the causative organism was not identified bacteriologically. A third patient was found to be infected with paratyphoid A and improved immediately on the use of vaccine.

The second division consists of a brief discussion of the enterococcus, in relation to a paper by Heitz-Boyer published in 1922.

The third and main part of the paper contains the report of a series of twelve cases of iridocyclitis and conjunctivitis due to *B. dysenteriae* Hiss.

Two types of ocular complications have been observed—hyperemia of the conjunctiva and sclera, and plastic iritis.

The scleroconjunctivitis of dysentery closely resembles metastatic gonorrheal conjunctivitis. It comes on rapidly, is intense, and subsides in from twenty-four to twenty-eight hours under ordinary treatment.

The iritis is of the common plastic type with photophobia, orbital pain, rapid loss of vision, ciliary congestion and swelling of the iris with a tendency toward synechiae. Posterior corneal deposits and vitreous opacity often reveal the involvement of the ciliary body and choroid. Hypotonia is the rule. Hypertension has not been observed.

These symptoms come on from twenty to thirty days after an intestinal upset. They may come on independently or they may be associated. Recurrences are common. They may be the only metastatic phases observed in the disease. As a rule, however, the ocular disturbances appear at the height of joint, urethral and general symptoms.

In all the cases reported, the serum of the patient agglutinated the bacillus of Hiss — up to a 1 : 600 dilution in some instances.

Specific antitoxin was used in some of the cases, with great benefit.

In closing, the authors call attention to the importance of giving attention to the intestinal tract in operative cases and cite an experience.

S. B. MARLOW.

CAROTID CAVERNOUS SINUS ANEURYSM WITHOUT PULSATING EXOPHTHALMOS. L. L. VILLORIA, Arch. d'ophth. **46**:157 (March) 1929.

The diversity of symptoms in cases of arteriovenous aneurysm involving the cavernous sinus prompts Villoria to record a case that did not show pulsating exophthalmos.

The patient was struck on the right cheek about four months prior to observation. He consulted an ophthalmologist because the right eye was blind and because of roaring in the head. The main features of the examination were a depression on the right side of the face, a blind right eye which was deviated down and in and was immobile except that when the patient tried to look to the right, it rotated inward. Tension in both eyes were normal. Roentgen studies showed evidences of flattening of the optic canal, obliteration of the sphenoidal and sphenomaxillary foramina, fracture of the lesser wing of the sphenoid and a marked increased density of the right orbit. Clinical examination showed a difference in the blood pressure on the two sides and a difference in the time of the cardiac cycle on the two sides, as measured by the polygraph.

The right internal carotid artery was ligated after digital compression had been applied intermittently for twelve days. It resulted in complete relief of the symptoms.

The author thinks that the blow displaced the lesser wing of the sphenoid *en masse*, closing the sphenoidal foramina and crushing the intervening structures. It thus prevented the occurrence of pulsating exophthalmos and other circulatory phenomena. The movement of the right eye to the left when the patient attempted to look to the right is difficult to explain. The difference in the blood pressure and the time of the cardiac cycle is explained by the presence of a large arteriovenous communication.

The presence of a normal intra-ocular tension in the right eye in this case leads the author to support the idea of a disturbed vascular equilibrium proposed by Velter to explain the hypertension in another case showing arteriovenous aneurysm.

S. B. MARLOW.

Injuries

COPPER IN THE EYE. H. COPPEZ, Arch. d'ophth. **45**:609 (Oct.) 1928.

Coppez reports three cases of copper in the eye. The first had been observed for twenty-four years. A penetrating wound in the ciliary region of the left eye resulted in a severe inflammation. Later, a cataract developed and was

removed. Sixteen years later, some thick capsule was removed for cosmetic reasons. There had also been a detachment of the retina, with loss of the upper half of the field of vision. After another eight years (twenty-four years after the accident), the patient reported muscae before the right eye. Examination gave negative results. At this time, complete examination of the left eye showed scattered patches of choroidal atrophy. There was no detachment of the retina, but no return in the field of vision. Roentgen examination revealed a foreign body in the anterior portion of the eye. The iris of this eye was bluish-green, and in the fundus were bluish patches of exudate. The author mentions the cases described by Priestly Smith, Goldzieher and Ertl which had been observed for much shorter periods. Haab's case was similar to that of the author, and the patient had been under observation an equal length of time. His second case had a less fortunate outcome. Enucleation was performed about six months after the accident. Pathologic study of this eye revealed the presence of two pieces of copper. The exudate present about them was greenish. The exudate filling the anterior chamber was also colored, particularly in the iris angle. The lesions found explain the bronzing of the cornea described by Jess. The copper salts were found precipitated between the endothelium and Descemet's membrane. In the third case, after the immediate reaction to the injury had subsided, it was possible to make out with the ophthalmoscope the characteristic greenish color of the retina. The foreign body was also visible. Later the eye was enucleated. Histologic study showed extensive impregnation of the retina.

S. B. MARLOW.

Operations

SYNDESMOPLASTY OF THE CORNEA FOR THE WEARING OF A PROSTHESIS. K. W. MAJEWSKI, Arch. d'ophth. **45**:673 (Nov.) 1918.

Although many different types of operation for the production of a good stump after enucleation have been described, Majewski points out that the defective eye of a one-eyed person is by far the best for the wearing of a prosthesis. Certain conditions are necessary before this can obtain. First, the blind eye must be somewhat smaller than the good eye. Second, it must be free from tenderness on pressure and other signs of irritation. Finally, the condition of the cornea is most important. The procedure described was evolved to overcome difficulties in this connection. The purpose of the operation is to cover the entire cornea with a layer of conjunctiva which is sutured over it after the epithelium has been denuded. The author has performed this operation successfully in a number of patients. It results in greater mobility of the prosthesis and does away with the sunken appearance of the upper lid so often present following other types of operation. Photographs show excellent results.

S. B. MARLOW.

INTRACORNEAL INJECTIONS. J. ROLLET, Arch. d'ophth. **45**:681 (Nov.) 1928.

The author first describes in detail the method he has used. Accidents rarely happen. A small scar where the puncture is made remains permanently. The method has been used experimentally to study the reaction of a large number of substances which are not detailed for lack of space. Therapeutic injections have been made in such a small number of cases that the author can only say that so far they have been favorable. The method may be used as a substitute for tattooing for extensive leukomas. A case is described in which the result was satisfactory. The method is simple to carry out if the proper attention is given to the details of execution. The author thinks that it will prove useful in three types of work outlined.

S. B. MARLOW.

Therapeutics

THE USE OF FOREIGN PROTEINS IN THE TREATMENT OF EYE DISEASE. W. L. BENEDICT and C. W. RUCKER, New Orleans M. & S. J. **81**:782 (May) 1929.

When proteins are used for their therapeutic effect in diseases of the eye, the specific effect is not essential. Only the cells in the vicinity of the lesion are sensitized to the invading organism, and they do not throw off enough antibodies to bring about healing. Under favorable circumstances, the production or exfoliation of a large number of antibodies of another toxin when thrown into the blood stream will augment the action of whatever specific antibodies are already present and will help to turn the indolent or nonhealing lesion into a healing one or at least will help to retard its progress. It is on the basis of this phenomenon that foreign proteins are used in ocular therapeutics and that the choice of proteins which may be used is not limited. The substances used in the cases referred to here are milk, typhoid vaccine and diphtheria antitoxin. Milk was most frequently chosen for injection because of the freedom from severe reaction or anaphylaxis. In primary uveitis, the results were not uniformly good. The best results were obtained in cases of iritis of short duration. In secondary uveitis after operations the results were good in 75 per cent of the cases. The influence was less in both primary and secondary form if the exciting cause persisted. The most satisfactory results were those obtained in cases of scleritis which had existed for months with constant pain. Redness clears up after two injections of 10 cc. of milk. Exudative choroiditis of obscure origin may show improvement when treatment with other remedies has been without effect. In penetrating wounds of the eyeball without retained foreign bodies, if the eye is not infected at the time of the injury, the immediate effect is to allay pain and reduce congestion. The authors conclude from their experience with patients treated with various proteins that the age of the patient has no bearing on the result of the treatment and that boiled milk is a safe protein to use. No anaphylaxis has been observed; some patients fail to react to injections of one protein. Other proteins may then be used. Large doses of diphtheria antitoxin can be given daily for from twenty to thirty days; some chronic diseases of the eye respond to protein therapy; the treatment should be continued long enough with sufficient quantity of protein to determine whether or not there is any effect. Protein therapy is an adjunct to general and local treatment but has great value in early inflammatory disease of the eye and should be used freely in hospitalized patients. Its value is considerably less for ambulatory patients.

W. ZENTMAYER.

THE INTRAVENOUS USE OF TYPHOID-PARATYPHOID VACCINES IN EYE DISEASES. H. J. HOWARD, Am. J. Ophth. **11**:685 (Sept.) 1928.

The clinical data on which this paper is based were obtained in Peiping over a period of three years. The author considers the use of typhoid-paratyphoid vaccine indicated in all acute and subacute inflammations of the eye, with the exception of those caused by the tubercle bacillus. It should also be given as a prophylactic measure against intra-ocular infection in penetrating wounds. He considers that given intravenously it is one of the best proteins for nonspecific therapy in many ocular disorders. The first dose should not contain more than twenty-five million bacilli for an average adult of good vitality. The therapeutic effect seemed to be proportional to the height of the fever induced. It is contra-

indicated in cases showing fever, low vitality and organic heart disease. It should not be used as a substitute for, but as an adjunct to, other effective means of treatment.

W. ZENTMAYER.

BACTERIAL ANTIGEN IN UVEITIS. R. E. MASON, Am. J. Ophth. **11**:702 (Sept.) 1928.

To avoid the severe shock that frequently follows the injection of a foreign protein, Mason employed an antigen, which contained *Streptococcus hemolyticus* with *S. aureus* and *albus* and which has a low protein content and is nontoxic. It was given intramuscularly in a dosage of 0.5 cc. every twenty-four hours or every second day. The maximum dose was rarely more than 2 cc. Eight patients, most of whom had acute uveitis, were treated, with such striking results that the author is of the opinion that it is specific in its action in this class of cases.

W. ZENTMAYER.

GENTIAN VIOLET IN OPHTHALMOLOGY. R. J. SISSON, Am. J. Ophth. **11**:893 (Nov.) 1928.

Experiments with cultures of *Staphylococcus albus* on laboratory mediums seemed to indicate that gentian violet and acriflavine hydrochloride might be distinctly valuable, especially in beginning intra-ocular infection, but the clinical results in six cases of intra-ocular infection with hypopyon due to a penetrating wound showed them to be ineffective, though there was a temporary disappearance of the hypopyon. Injections of the dyes were made into the anterior part of the vitreous in dilutions of from 1 : 5,000 to 1 : 15,000 after evacuation of the anterior chamber.

W. ZENTMAYER.

VASCULARIZATION OF THE CORNEA STIMULATED BY LINEAR CAUTERIZATION; FILARIA IN THE EYE. F. SAEGER, Klin. Monatsbl. f. Augenh. **82**:223 (Feb.) 1929.

The author asserts the necessity of stimulating the formation of blood vessels in the cornea under certain conditions. Blood vessels will form spontaneously as a protection of the cornea, which normally contains none, in parenchymatous keratitis and other conditions. This indicates the beginning of repair. Trachomatous pannus, on the other hand, seems to be part and parcel of the granulation tissue invading the cornea. An attempt is made to curb this excess development. In serpent ulcer, again, and in other purulent forms of keratitis, the toxic condition may prevent the formation of blood vessels. The same observation is made in indolent forms for unknown reasons.

Vivid formation of blood vessels may be produced by drawing, with the loop of the galvanocautery, a line from the conjunctiva across the limbus into the infiltrated zone. Blood vessels will form along this line, and in a comparatively short time they will arrive at, and spread within, the infiltrated area. The shortest radius should be chosen, and optically important areas should be avoided. Similar considerations led Elsching to use the galvanocautery in keratoconus. Initial failures were turned into good results as soon as the cauterization was extended into the limbus.

One cauterization of this description yielded a prompter result than repeated cauterizations of the center of the cornea. Elsching recommends the use of a slightly glowing platinum loop which will insure slow desquamation and partial organization of the cauterized tissues.

Two cases are reported. In the first, a splinter of stone protruded into the anterior chamber. It was surrounded by an infected ulcer of the cornea; the exudation extended to the anterior lens capsule; hypopyon and iritis were present; the fundus could not be seen. The xerosis bacillus and gram-positive diplococci were found. Numerous recurrences of the hypopyon occurred, as the foreign body was left in situ because it was feared that an attempt to remove it would spread the infection into the globe. Linear cauterization was done, extending from the conjunctiva, across the limbus and into the infiltration. Vessels began to form along this line four days later, causing the spontaneous freeing of the foreign body. It dropped to the bottom of the anterior chamber, where it remained. The eye was free from irritation, and vision was 6/8 when the patient was seen again a month after he had returned to work. No mention was made of the fate of the eye after December, 1928. In connection with this case, the author warns against the removal of splinters of iron during the active infections. He considers it safe to postpone the extraction until beginning siderosis may be found by the use of the slit-lamp, or when diminution of the dark-adaptation has developed.

The second case gave a history of infestation with *Filaria loa*, dating back to 1912 when the patient had lived in Kamerun. The right eye had been periodically inflamed since that time, more frequently since 1921 and constantly since October, 1927. It had been treated for four months prior to the time when the author saw the patient in March, 1928. There were: a large corneal infiltration with hypopyon, wavy Descemet's membrane, stippled epithelium and diminished sensitiveness of the cornea. Fluorescein stain was vivid. Numerous recurrences of the hypopyon had occurred; the usual therapy, including curetting of the cornea, had failed. Linear cauterization, similar to that in the first case, was followed by recovery within two weeks. The result was permanent and the eye free from irritation and pathologic changes, except a paracentral leukoma in October, 1928. The presence of the worm in the globe itself, syphilis and corneal herpes could be excluded.

K. L. Stoll.

Comparative Ophthalmology

The Question of Visual Capacity in Mice Bearing Rodless Retinas. Clyde E. Keeler, Ztschr. Vergleich. Physiol. **7**:736, 1928.

The author has reported elsewhere the results of experiments on a breed of mice which did not possess rods in the retinas, which led to the conclusion that these animals have no vision. This view has been challenged by Hopkins, who worked on the same strain of animals. In this paper, Keeler produces further evidence of the absence of visual function. He shows that no photo-electric changes are detectable by the method of Chaffee and Hampson in the retinas of these mice when exposed to light.

Francis Heed Adler.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

DR. THOMAS H. JOHNSON, *Secretary*

Meeting, April 15, 1929

DR. BERNARD SAMUELS, *Chairman*

PRESENTATION OF A CASE OF HEREDITARY NYSTAGMUS. DR. WILLIS S. KNIGHTON.

HEMORRHAGES IN THE FUNDI IN HYPERTENSION. DR. FRITZ LANGE, Munich, Germany.

These papers will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. MARK J. SCHOENBERG: I should like to ask whether Dr. Lange is familiar with, or has tested out, the work of Horniker. This author has taken the blood pressure in both arms at the same time in about 1,000 cases, and in some cases he found differences of from 17 to 50 points. According to his experience, the side which presented the conjunctival and retinal hemorrhages corresponded to the side on which the blood pressure was highest.

DR. MARTIN COHEN: We have observed cases of hypertension for ten years or more, and the patients with pure hypertension, or essential hypertension, have never shown any vascular changes ophthalmoscopically, except in two cases in which arteriosclerotic changes did develop, going on to what is known as arteriosclerotic retinitis. One patient with a condition of the eye died and autopsy showed marked arteriosclerotic changes in the choroid. It is questionable in my mind whether some of Dr. Lange's cases of pure hypertension might not be classed as cases of early changes of the vascular wall, as he mentioned retinal changes which do occur in lesions of the vascular wall, such as changes in caliber and diminution in the lumen of the vessels. Clinically, these observations would be considered incipient lesions of the vascular wall and not cases of pure hypertension. It is frequently difficult for an ophthalmologist to determine which are the pure vascular cases, as a complete physical examination is essential. The internist should establish the cases which are to be considered purely hypertensive. I have recently seen a case of retinal hemorrhages in which the examination of the heart, chemical analysis of the blood and examination of the other organs of the body did not reveal hypertension. I believe that the classification of retinal hemorrhages as belonging purely or essentially to cases of pure hypertension is, for the present, practically impossible.

I should like to ask Dr. Lange whether he has studied the intravascular pressure in any of his cases, or whether he knows if any such study has been conducted in the cases of hypertension, because I know from reports in the literature that the

ocular intravascular pressure may be normal or even below normal in cases of general hypertension.

Has Dr. Lange found that in the combined cases of arteriosclerosis plus hypertension, the condition exists unilaterally or bilaterally? Most of the cases of so-called arteriosclerotic retinitis associated with hypertension that lasted for six months or more have been unilateral rather than bilateral. I do not know an established reason for this, unless it is considered a local vascular manifestation. The entire subject of differentiation between arteriosclerosis and hypertension is still clinically debatable. Some authorities are of the opinion that hypertension is a forerunner of pure arteriosclerosis. It is possible that unless more definite clinical evidence is established, one will be unable to make a definite differentiation of the fundus with respect to this subject.

DR. SIGMUND AGATSTON: I am rather interested in this subject, and have done some work along these lines, and I agree with Dr. Lange that it is impossible to get hypertension, even essential hypertension, without arteriosclerosis or some changes in the fundus. These changes I am sure, have not been recognized by everyone in the past. I do not think that it is necessary to go to internists for information. I think that we ophthalmologists have a vast amount of information concerning the fundus which the internist can use. We know much more about arterioles by examining the fundus carefully than the internist knows. In my consultations at the hospitals I feel that I learn more about the patient's arterial system by looking at the fundus than the internist does by his careful laboratory methods. I think that it is impossible to have a hypertension of 200 without some change in the fundus. The changes are distinct, if one knows where to look for them. The angiospasm which is supposed to be responsible for hypertension can be seen definitely in the fundus, if one looks for it. Some of the branches of the arteries become narrower, and paler in spots, and show contractions; these may be transient or they may be permanent, and the arteriosclerosis is a secondary arteriosclerosis. The disease was originally angiospasm, and the hemorrhages and other changes are secondary. Aside from that, I have found that it is impossible to have hypertension without the indentation of the vein. That is not a sign of arteriosclerosis, but a sign of increased blood pressure. If one looks at the fundus one can evaluate the blood pressure by the amount of pressure of the artery on the vein as it passes over it. It is impossible to have high blood pressure without that indentation. Many cases of arteriosclerosis in one eye are purely secondary arteriosclerosis brought about by spasm which is persistent, and which is the result of partial or complete occlusion of the artery. I am collecting a number of cases of this type. It is a vast subject, and one in which I am much interested, and I hope to be able to say more about it in the future.

DR. FRITZ LANGE: In reply to Dr. Schoenberg's question, I should say that I know the work of Horniker, but have had no experience as to whether conjunctival hemorrhage occurred on the side in which the blood pressure was highest in the arm. I have seen only a few cases of conjunctival hemorrhages, and I have made no observations on this point.

What Dr. Cohen says about the localization of these changes in the eye is interesting. It makes one wonder why there are no changes in the eye in so many cases of hypertension, and, on the other hand, why changes do occur in many cases. One must believe that hypertension is not a single disease. I think that until now many different diseases were grouped together, and perhaps later on we shall be able to separate them. It is known that hypertension may occur in different parts of the circulatory system. If these patients are examined clinically,

it is found that one claims he has headaches, another has trouble with the heart, a third has trouble with the kidneys and so on. The blood vessels are principally affected, but they may be affected in different regions of the body in this disease, so that in one case the blood vessels in the fundus of the eye are involved, and in another case they are not. Volhard made the distinction of pure essential hypertension in the benign form, and in the malignant form, and those cases in which one sees hemorrhages in the background of the eye are really not benign cases. In these cases the prognosis is worse than in others in which there is no change in the eye, and one finds that the changes in the eye are often combined with changes in the kidneys. Then again, in other early cases one often finds no disease of the kidneys, but a disease of the fundus.

Answering the question about arteriosclerosis, I should say that there are two kinds, the first being arteriosclerosis following hypertension, which means that one at first finds hypertension without any signs of arteriosclerosis, but later on arteriosclerosis appears, and the second, pure arteriosclerosis, embracing cases which never had and never will have hypertension. In the clinic, cases of pure arteriosclerosis were followed for a long time, and patients whose cases were followed for ten or fifteen years did not show hypertension at any time during that period, although the arteries showed signs of calcification. I do not think that such cases of pure arteriosclerosis have anything to do with hypertension.

In regard to the question whether one finds such changes predominant in one eye, I cannot answer. I found them in one or both eyes. In the Rockefeller Hospital, I have two cases in patients, aged 19 and 22, without arteriosclerosis, and they have marked changes in both eyes. I cannot say that the change is predominant in one eye.

In regard to what was said about the collaboration of internists and ophthalmologists, there is no doubt that there must be much more cooperation than there has been. There is really so much for the internist to see in the fundus of the eye, and that is not all—the ophthalmologist has to learn much more about the fundus of the eye.

OCULAR PATHOLOGY OF PERIARTERITIS NODOSA. DR. ISADORE GOLDSTEIN and DR. DAVID WEXLER.

This paper appears in full in this issue, p. 288.

DISCUSSION

DR. MARK J. SCHOENBERG: I recall that six or seven years ago I saw a patient in the Presbyterian Hospital with a condition that was diagnosed periarteritis nodosa. This patient, a woman, aged about 25, suddenly became blind in one eye; I was called to look at the fundus. The good eye showed nothing abnormal, and the other eye had the characteristic picture of acute obstruction of the central retinal artery.

TWO CASES OF PLASTIC REPAIR BY THE USE OF A LINED PEDICLE GRAFT FOR CLOSING A HOLE FROM THE ORBIT INTO THE NOSE. DR. WENDEL HUGHES.

CASE 1.—A man had an exenteration of the orbit two years before presentation which left a fistulous opening into the nose. A pocket was made just inside the upper orbital margin in the skin which was formerly the upper lid into which was inserted a flat form of dental compound wrapped in a layer of epidermis from the leg, of tissue paper thickness, with the raw side externally. The superficial layer

grew to the skin and formed a lining for the part that was to be turned down into the opening so that it would be buried and would cover the end of the inner surface of the pedicle that was to be turned down to cover the fistula into the nose. The next step was to turn this lined pedicle down. This was done two weeks later, the grafted skin on the inside being trimmed to fit the opening. The part left by the removal of the pedicle was already covered by the deeper layer of skin which formed the lining of the pocket formed by the first procedure. One month later the pedicle was cut and the edges were trimmed. The result was complete closure of the fistula.

Case 2.—A woman had a fistula into the side of the nose following a radical external operation on the ethmoid and frontal sinus which was repaired in a manner similar to that in case 1; the pocket was made in the upper lid and the pedicle was swung down from there.

The idea for this procedure was obtained from a procedure originally carried out by Dr. J. M. Wheeler for a similar condition, a report of which has not been published.

Chronic Conjunctivitis and Its Treatment. Dr. H. Birnbaum, Antwerp, Belgium.

The results in chronic conjunctivtis are often not successful for two reasons; the disease, as such, and the remedies.

Chronic conjunctivitis often has causes which are more profound. It is often caused by external agents, or it accompanies old processes of the lids and their margins. In these cases, collyria cannot help much. In other cases, the chronic conjunctivitis is an extension of a chronic rhinitis or processes in the nose; these cases are frequent, and the condition of the nose must also be investigated.

A subacute conjunctivitis frequently is a sequel to a recent postnasal catarrh, and clears up without treatment just as nasal catarrh does, however, a chronic conjunctivitis following a chronic rhinitis is more tenacious, and one cannot help much with collyria.

An example of conjunctivitis due to a disease of the whole body is the conjunctivitis eczematosa of children with scrofula. I am sure that there are cases of conjunctivitis due to gout. I know of a woman who lost one and a half of the ovaries in an operation. Her eyes become red every month when the menses, which are reduced, approach.

The remedies are chiefly sulphate of zinc and nitrate of silver. Sulphate of zinc is said to be a good astringent. It is, but in strong solutions it is caustic and keeps up an irritation. In weak solutions its action is of short duration and is palliative like epinephrine. The second chief remedy is nitrate of silver. It is known also that nitrate of silver provokes a secretion which is the result of the chemical action of silver preparations. I use a strong silver preparation in acute cases in which there is purulent secretion, but I see no utility in its employment in chronic cases, if not in a 0.25 per cent solution, or in cases in which there is slight secretion in the morning.

Before all treatment, however, one must ascertain if there is any abnormal condition in the nose. In these cases, if I give sulphate of zinc, I recommend that my patients touch the membrane of the nose with the same collyrium.

There is, on the other hand, blepharoconjunctivitis, which cannot be healed without probing and dilatation of the lacrimal duct.

When I want to employ a stronger remedy, I take a pencil of copper sulphate and touch the conjunctiva with it, as in trachoma. The action of this strong

remedy can be graduated; one can touch the conjunctiva strongly or gently, so gently that the sensation of burning does not last for more than a few minutes; then one can touch the points of the conjunctiva, which have been chosen as many times as necessary. I am sure that the pain after the application of copper sulphate, if the conjunctiva is touched gently, is not as great as that after the use of a strong solution of silver. In many cases, it is sufficient to repeat this treatment four or five times during two or three weeks.

There are cases of chronic conjunctivitis in which there is more redness than annoyance, and in which the conjunctiva becomes red quickly if anyone smokes in the room. There is probably a great sensitiveness of the vessels of the conjunctiva. In many of these cases, redness becomes permanent and one has great trouble to relieve it. For such cases, I use alum, and not infrequently it helps when other remedies fail. The sensation of burning lasts for half a minute, and after the treatment the patient feels relieved and the eyes become pale. This treatment must be repeated once or twice a week for a long time.

I am partial to massage. One takes the two margins of the lids between the two fingers and massages them. A glass rod with a ball at its end is placed under the upper lid or the under lid, and the lid with the conjunctiva is pressed against the finger, with more or less force.

A preparation (*Ann. d'ocul.*, February, 1929) was used, which contained silver and tannin, or, more exactly, it was composed of albuminate of silver, and diacetyltannin. The preparation contains 6 per cent silver; nevertheless, in this composition the silver does not precipitate albumin. The chief advantage of the preparation is that the tissue is penetrated and becomes a bad ground for the growth of microbes.

After its application one sees no secretion like that found after the use of nitrate of silver or strong silver protein. Its astringent action lasts for about twelve hours. In acute cases it does not last as long. In all typical cases of conjunctivitis, acute or chronic, it causes great relief and its application gives no pain. I use it in a solution of 5 per cent. The solution was first prepared by two physicians in Breslau, Siebert and Henry Cohn.

DERMATOSES OF THE REGION OF THE EYE. DR. CHARLES MALLORY WILLIAMS.

This paper will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. RAYMOND E. MEEK: A number of years ago, Verhoeff, and also Hamburger made an observation in regard to vascular nevi around the eye and the occurrence of glaucoma on that side. I should like to ask Dr. Williams if he has ever noticed the occurrence of glaucoma on that side of the face in which a nevus occurred.

I saw a woman last week who had secondary syphilis and who was treated with six injections of 0.9 mg. of neoarsphenamine. An arsenical dermatitis developed following the treatment. I was called in to see her because she had pain in the eyes, and I noted a punctate keratitis on the right cornea. The condition almost resembled dendritic keratitis. The left eye had the same changes with the punctate lesions and abrasions of the cornea. I should like to know if these changes are seen in arsenical dermatitis, or if they are an accompaniment of secondary syphilis, or any other dermatitis.

DR. CHARLES MALLORY WILLIAMS: With regard to the first question, I know of no connection between nevus and glaucoma. It is hard for me to believe that

there should be a connection. Nevi are exceedingly common, and there is no reason why any other disease should not occur in connection with them. There is no reason why they should not occur in the same patient, but I have not seen such an occurrence, probably because I never see glaucoma.

In regard to the punctate keratitis, I have not seen that condition in secondary syphilis or after the use of arsenic. Of the two, I should think that arsenic was the more likely cause for the condition. Arsenic will produce a large variety of lesions. Arsphenamine may cause an exfoliative dermatitis, and I have seen lesions exactly resembling zoster, and I think that these punctate lesions might resemble the small vesicles which may occur in a zoster-form eruption, but that is pure guess-work. I have never seen any cases of that type.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

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April 19, 1929

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Thiersch Graft for Symblepharon. Dr. T. B. Holloway.

Dr. Holloway exhibited a patient on whom he had operated for a symblepharon, produced as the result of a lime burn. The palpebral attachment of the adhesion occupied about 8 mm. in the central part of the lower lid, and extended to the margin; several bands crossed the fornix and were attached to the bulbar conjunctiva and the lower portion of the cornea. The last named structure showed marked and complete vascularization.

Both extremities of the symblepharon were dissected free, and the culdesac was deepened. The lateral portions of the bulbar conjunctiva were then undermined and the margins sutured so as to bring a flap over the cornea for about 1 to 2 mm. A piece of Stent's composition about 5 mm. wide had been previously molded to conform in shape with the contour of the lower culdesac and almost corresponding in length to that of the lower lid. Two holes were then placed in each end of the stent, large enough to permit the passage of a needle. A Thiersch graft was taken from the thigh, and this was shaped about the stent, the latter was sutured in the lower culdesac by means of a mattress suture at each end, which passed directly through the whole thickness of the lower lid. The loop of the mattress suture was placed between the inner surface of the stent and the outer surface of the inner fold of the graft.

In this case, as in a previous one, an excellent result was obtained.

A Case of Retinitis Punctata Albescens. Dr. L. F. Appleman.

A young woman, aged 20, of Jewish parentage, had retinitis punctata albescens, which had been present since childhood and was probably congenital. The chief symptoms were night blindness, with severe headaches, which were aggravated by the close clerical work that she did. Vision in the right eye was 6/9—1; in the left, 6/9—4. This was improved to 6/6 in each eye by plus 2.25 diopters spherical lens plus 1 cylinder axis 75 degrees in the right, and plus 1.50 cylinder axis 80 degrees in the left.

The visual fields showed a slight concentric contraction for form, which was increased when they were taken under reduced illumination. The color fields were greatly contracted.

The results of external examination were negative.

Ophthalmoscopic examination revealed hundreds of white, rounded, nonpigmented spots in the deeper layers of the retina and dispersed throughout the whole eyeground in each eye. Both maculae were free from the spots. The spots were unaccompanied by any pigmentary changes and there had not been any progressive loss of sight. These facts, together with the symmetrical distribution of the spots, point to the congenital nature of the condition.

The blood and the urine were normal.

Examination of two sisters and one brother of the patient revealed no ocular abnormality. The father and the mother of the patient were first cousins. This fact has been commented on in previous reports of cases and in the description of the condition.

Dr. Alfred Cowan made good photographs of the condition in this case.

DISCUSSION

Dr. Holloway stated that it had been his privilege to see probably more than his share of retinal cases belonging to this group, owing to his long service at the Pennsylvania Institution for the Instruction of the Blind at Overbrook. He said that this was certainly the most typical case of this kind he had seen, and that if any of the younger men had failed to see it, he hoped they would request Dr. Appleman to give them the privilege of examining the patient at some other time and place.

RECIPROCAL INNERVATION OF THE EXTRA-OCULAR MUSCLES IN A CASE OF BILATERAL PARALYSIS. DR. F. H. ADLER.

A case of complete bilateral oculomotor paralysis showed the following: On extreme abduction to the right, not only did the right eye turn out, innervated by the right external rectus, but the left eye turned in slightly toward the midline. On looking to the left, the left eye could be voluntarily abducted and the right eye followed inward slightly toward the midline. There was no evidence of any power in any of the external ocular muscles save the external rectus on each side; therefore, the movement inward of either eye must have taken place by a relaxation of the tone of the external rectus muscle. This relaxation of tone in one muscle when its antagonist is innervated constitutes the well known reciprocal innervation first described by Sherrington. The paper will appear later in full in the ARCHIVES.

GLUTATHIONE IN THE CRYSTALLINE LENS. DR. I. S. TASSMAN and DR. W. G. KARR.

Employing the nitroprusside reaction as a guide, some of the earlier investigators concluded that the amino-acid cysteine was present in the normal crystalline lens and absent in the cataractous lens. Heffter later described the oxidation-reduction process of cystine, due to the activity of its labile hydrogen atom of the SH radicle present in its molecule, and pointed out its possible importance as a factor in cell metabolism in the tissues. The substance had not, however, been demonstrated in the tissues in its free state. In 1921, Hopkins discovered glutathione, which he described as a combination of cystine and glutamic acid, and after producing it from fresh yeast, he also showed that it was present in the liver and other organs. Glutathione also showed the same oxidation-reduction property in the tissues and was considered necessary in the proper functioning of the cell. Hopkins estimated

glutathione in the liver of the rat to the amount of 0.18 per cent; in the liver of the rabbit to the amount of 0.24 per cent. This was greater than the percentage found in other organs. Michail found it to be present in the lens of oxen, rabbits and dogs to an average amount of 0.356 per cent. This exceeded the percentage found by Hopkins in the liver.

In our own investigation, we used freshly extracted pig lens from animals of uniform age and weight, and after the percentage of dry matter was determined (31.35), the percentage of glutathione was estimated, in a series of seven experiments, to be on the average 0.296 (according to the Perlzwig and Delrue modification of the Tunnicliffe method). The total cystine obtained (Folin and Looney method) was on the average, 1 per cent. The cystine percentage was therefore 0.704, giving an average equilibrium ratio of cysteine to cystine of 0.420. It was also found that the process of desiccation reduced the percentage of glutathione in the normal pig lens to 0.224 per cent in a period of from three to four weeks. A sample of fresh lens extracted with 5 per cent trichloracetic acid and allowed to stand at room temperature for twenty-four hours, showed a reduction of glutathione to 0.106 per cent.

In the freshly extracted human cataractous lens, the content of dry matter was found to be higher than in the normal pig lens, averaging 35.5 per cent. In an attempt to estimate glutathione in the cataractous lens, it was found that there was practically none present. The total cystine in cataractous lenses was estimated at 0.8 per cent, which was lower than the similar content in normal pig lens. This total, however, represented all cystine, since it was found that there was no reduced glutathione present.

From these results, it is seen that glutathione is present in the normal pig lens in definite amount. An equilibrium ratio was established also between cysteine and cystine. In human cataractous lenses, glutathione could not be found in sufficient quantity to estimate. The cystine percentage obtained in human cataractous lens was 0.8.

The effect of various influences on the oxidation-reduction of cysteine and glutathione have been described from time to time. Thompson and Voegtlin, in studying glutathione, felt that the toxic action of certain substances is due to a primary disturbance of the cellular oxidation-reduction process, resulting from a chemical interaction of the poison with the glutathione of the cell. In a long series of experiments, they estimated the percentage of glutathione in the liver of rats at 0.183 per cent (according to Tunnicliffe's method).

Future studies of glutathione and the development of methods for the measurement of the oxidation-reduction potential in the tissues may add more to the knowledge of the crystalline lens and its change from the normal to the abnormal state.

THE RÔLE OF CALCIUM IN ESSENTIAL GLAUCOMA. DR. JOSEPH I. GOUTERMAN.

Experimental, clinical and therapeutic evidence was submitted, suggesting two main premises and their possible intimate relationship: (1) a prevailing sympathetic hyperexcitability in essential glaucoma, brought about by (2) a calcium disturbance. Calcium in the human blood was briefly discussed, with emphasis on the two forms of calcium—the nonionizable and the ionizable or electrolytic variety. The author favored the belief that the ionizable portion of calcium is diminished in glaucoma, the total calcium not necessarily being impoverished. The standard laboratory methods for the determination of the various forms of calcium were mentioned. The characteristic clinical features and their detection in cases of glaucoma were referred to. The paper was concluded with an outline of calcium therapy. Six cases, graphically illustrated, were reported.

Book Reviews

INFETIONEN DER CONJUNCTIVA. By PROF. T. AXENFELD, Freiburg. Reprint from Handbook der Pathogenen Mikroorganismen. Vol. 6. Pp. 281-354. Jena: Gustav Fischer and Berlin; Urban & Schwarzenburg, 1928.

The monograph takes up only a few of the principal infections of the conjunctiva. The entire subject will be reviewed in the second edition of the author's "Bakteriologie in der Augenheilkunde." A more or less new method for obtaining material in the study of conjunctival infections is Lindner's "epithelial scraping." The technic is carefully described, and mention is made of Pillat's investigations on the topography of the saprophytic conjunctival germs. Though the author recognizes the scientific value of this method, the diagnostic value of the old examination of a conjunctival smear when stained by Gram must still be maintained, especially as no new diseases excepting the inclusion-body diseases have been discovered by epithelial scraping, and the old method still remains the simplest and most practical for general use.

The conjunctivitis of the Koch-Weeks bacillus and of the influenza bacillus, pneumococcus conjunctivitis and diplobacillary conjunctivitis are exhaustively described, their distribution, clinical picture, epithelial scraping and smear examinations, culture, morphology, serodiagnosis, pathogenicity, diagnosis and bibliography being given. It is needless to say that the subject has been most carefully covered in a manner in keeping with the publications of their distinguished author. A. K.

DIE RADIUMBEHANDLUNG IN DER AUGENHEILKUNDE. By PROF. LEO KUMER, Professor of Dermatology in Innsbruck, and L. SALLMANN, M.D., Assistant in the II Universitäts Augenklinik, Vienna. Price, 14.40 marks. Pp. 198 with 66 text illustrations. Berlin: Julius Springer, 1929.

The purpose of this book is to instruct the oculist on radium therapy and is the outcome of many years' collective work between the eye clinic in Vienna, and the skin clinic, of which the radium institute is a part. The general part gives a physical introduction, a description of the technical part of radiation, and the action of radiant waves. In the clinical part, the radium treatment of malignant tumors is first taken up; then follow its application in diseases of the lids, conjunctiva, lacrimal organs, cornea, sclera, uvea, retina, lens, glaucoma, optic nerve, myopia, vitreous and orbit. A complete bibliography is appended.

The methods of applying radium are first described with its filtration and the precautions necessary to avoid undesired action and injury to the tissues. After discussing its biologic action on the eye, the injuries to the eye and its surroundings from radium are taken up (p. 37 to 48). The clinical part is the one which will interest the practicing ophthalmologist most, and the following are a few of the important conclusions arrived at by the authors. Radium therapy is generally the best treatment for angioma of the lids. Pigmented nevi should never be exposed

to radium, as radium cannot dispel the pigment. Carcinoma of the lids is suited for radiation as long as the tarsus or the bone is not involved; this method may at times be combined with surgery. Radiation plays an important rôle in the treatment of spring catarrh, though cases have not been observed long enough to determine a permanent cure. Radium furnishes a definite improvement in the treatment of epibulbar carcinoma by operation. The results with radiation to arrest lacrymation after tear-sac extirpation are not good. The authors are in favor of treating tuberculous iridocyclitis with radium especially when combined with tuberculin therapy. Radium treatment in glioma of the retina should be considered only in the case of the second eye when enucleation is refused. Prophylactic radiation of the orbit after enucleation for glioma even when there are no signs of extension of the primary tumor is now the general practice. Though cataract is a frequent form of radium injury to the eye, it is curious that attempts have been made to clear up beginning opacities in the lens with x-rays and radium. In the opinion of the authors, this is as mistaken an endeavor as the old and new nonoperative methods of treating cataract.

We owe the authors thanks for this timely publication. They have collected information of great importance to every ophthalmic practitioner. Their opinion on treatment with radium is fair and conservative and is based on a large personal experience.

A. K.

Directory of Ophthalmologic and Otolaryngologic Societies *

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INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

President: Dr. E. Treacher Collins, 17 Queen Anne St., London, England.
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Place: Amsterdam, The Netherlands. Time: Sept. 5-13, 1929.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

Chairman: Dr. T. B. Holloway, 1819 Chestnut St., Philadelphia.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Detroit. Time, 1930.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
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Place: Atlantic City, N. J. Time: Oct. 21-25, 1929.

AMERICAN OPHTHALMOLOGICAL SOCIETY

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Place: Richmond, Va. Time: June, 1930.

LOCAL

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President: Dr. Harold S. Muckleston, 6422 Hollywood Blvd., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Dr. Louis S. Greene, 1710 Rhode Island Ave. N. W., Washington.
Secretary: Dr. R. E. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. F. McDuffie, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Albert E. Bulson, 416 W. Berry St., Fort Wayne, Ind.
Secretary: Dr. Robert J. Masters, 601 Hume-Mansur Bldg., Indianapolis.
Place: French Lick Springs Hotel. Time: December 12 and 13, 1929.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Robert J. Masters, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. M. Earle Brown, Maison Blanche, New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., third Thursday of each month from September to June.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Leo J. Goldbach, 1012 N. Charles St., Baltimore.
Secretary: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

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President: Dr. W. Holbrook Lowell, 82 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Eye Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. N. Spratt, 900 Nicollet Ave., Minneapolis.
Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. R. Forgrave, Logan Bldg., St. Joseph, Mo.
Secretary: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. William H. Luedde, Metropolitan Bldg., St. Louis.
Secretary: Dr. Leo L. Mayer, Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Frederick J. Wort, 1080 Broad St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Robert M. Rogers, 680 St. John's Pl., Brooklyn.
Secretary: Dr. William F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. John P. O'Keefe, 251 State St., Albany.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. E. E. Blaauw, 190 Ashland Ave., Buffalo.
Secretary-Treasurer: Dr. A. F. Luhr, Medical Arts Bldg., Buffalo.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. E. F. Krug, 12 W. 44th St., New York.
Secretary: William F. C. Steinbugler, 815 Park Ave., New York.
Time: 8:30 p. m., third Monday of every month.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Cecil B. Hert, 29 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George M. Constans, Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Myron Metzenbaum, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7:00 p. m. Third Wednesday of each month from October to May.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Joseph L. McCool, 909 Stevens Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

THE PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

THE PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur Jones, 105 N. 8th St., Boise, Idaho.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. C. E. G. Shannon, 17th and Walnut Sts., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William Thornwall Davis, 927 Farragut Sq., Washington, D. C.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Miami, Fla. Time: Nov. 20-22, 1929.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. D. L. Bettison, Medical Arts Bldg., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, Medical Arts Bldg., Dallas, Texas.
Recording Secretary: Dr. L. A. Nelson, 4105 Live Oak, Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. P. Schenck, Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

HOUSTON ACADEMY OF MEDICINE, EYE EAR, NOSE AND THROAT SECTION

President: Dr. John H. Foster, 1300 Walker Ave., Houston, Texas.
Secretary: Dr. Edward W. Griffey, Houston Clinic, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

THE SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. H. Roseborough, 302 Moore Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Will Otto Bell, Medical-Dental Bldg., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Herschel Ezell, Doctor's Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. H. B. Stone, Roanoke.
Secretary-Treasurer: Dr. Fletcher D. Woodward, Box 162, University.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. O. M. Rott, Paulsen Bldg., Spokane Wash.
Secretary: Dr. C. A. Veasey, Jr., Spokane, Wash.

THE MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. L. P. Allen, 145½ Main St., Oshkosh, Wis.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.